Obstetrics A



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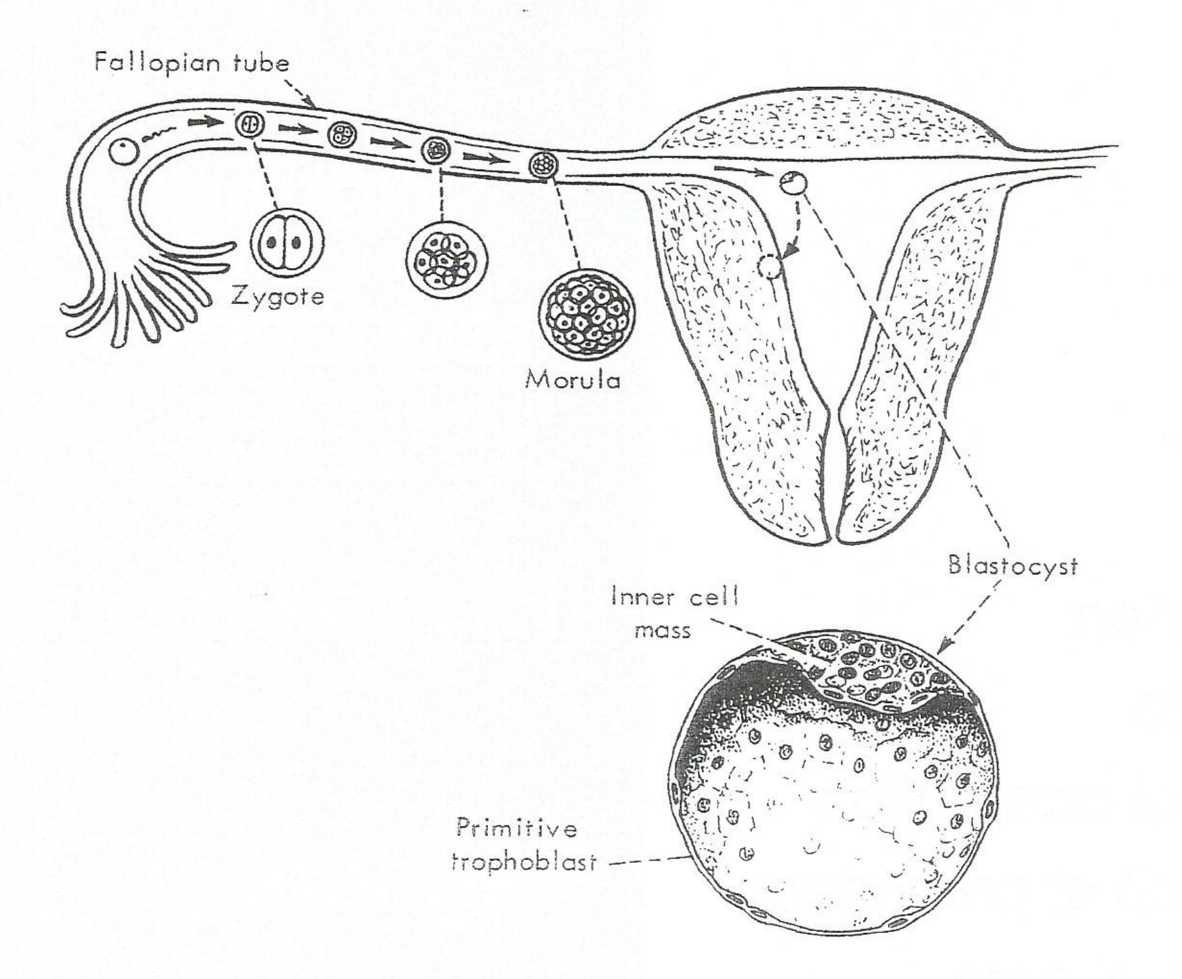
Chapter 1

Normal Pregnancy

(Physiology of Pregnancy)

- Fertilization
- Placenta
- Maternal adaptation
- Diagnosis of pregnancy
- Ante-natal care

OVULATION 12th Day
FERTILIZATION
Spermatozoon
Zygote
Progesterone
Oestrogen
Day of Cycle 5 10 15 20 25 30
Morula
Blastocyst
Embedded Blastocyst 26th Day
Repair Proliferation Secretory Decidua



Physiology of pregnancy

Fertilization

- **Definition**......The *union* of a mature *ovum* & a mature *spermatozoon* at ampulla of F.tube ¤ (bet. outer & middle ⅓) → zygot

- **Transport of sperms** movement occurs through the action of tail stimulated by PGs present in semen.
  - Mature sperms [22x or 22y] reach F.tube..........*within 40 min*
  - **Capacitation** of sperms starts........................*within the cx*

    some says that capacitation starts earlier in the epididymis.

    ↘ changes in sperms to ↑ its ability to fertilize

    Takes about 26h. . Removal of excess proteins → present in acrosomal cap.

    . Production of enzymes e.g. hyaluronidase ¤, collagenase.

- **Transport of ovum**
  - Ovulation → completion of meiosis I → 1^{ry} oocyte + 1^{st} polar body
  - **Ovum pick** up occurs by the tubal fimbria → then it is carried by ¤ (specially fimbria ovarica stimulated by PGs.)

    ↘ passive fluid currents (helped by ciliary action & peristalsis)

- **The Union**
  - **1** sperm penetrates the ZP (**Polyspermia** is prevented by: **Zonal block**) (thickening of Z.P (fertilization memb.).)
  - Meiosis II starts after fertilization (♂)

    ↘ formation of 2^{ry} oocyte (♀ pronucleus) + 2^{nd} polar body ¤

    ↘ Sperm head swells ¤ to form the ♂ pronucleus

Differentiation

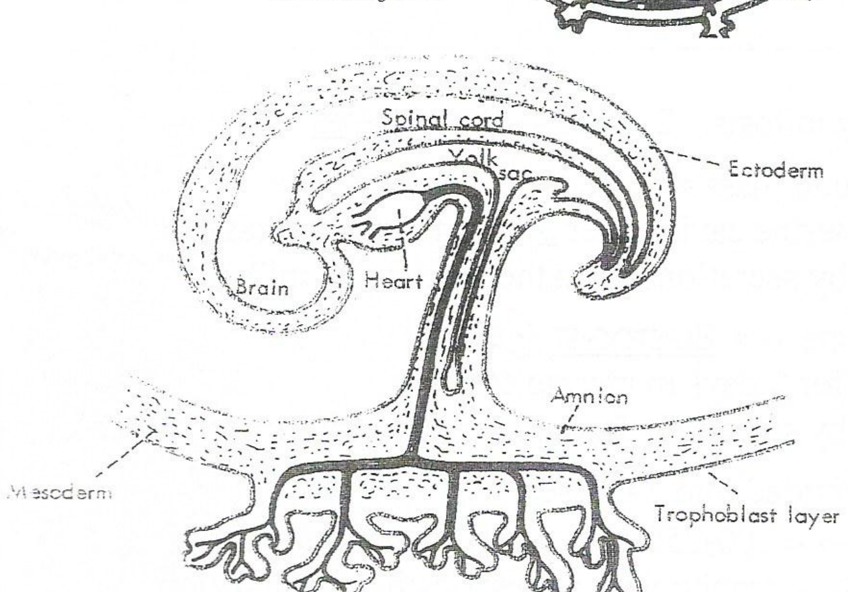
- Zygot rapidly divides by mitosis...2...4...8 → <u>BLASTOMERES</u>
- A <u>MORULA</u> is formed (round mass ≈ 16 cells)
  - It reaches the uterine cavity after <u>3</u> days from fertilization → delayed by utero-tubal sphincter
  - It is nourished by secretions from the tube (tubal milk)
- Fluid will then accumulate → a <u>BLASTOCYST</u> (chorion).
  - It remains free for <u>3</u> days in uterine cavity
  - It is nourished by secretions from the endometrium (uterine milk)
- The blastocyst will be divided into 2 masses:
  - <u>INNER CELL MASS</u> → ≈ 100-250 cells ⇒ will form the embryo
  - <u>OUTER CELL MASS</u> → trophoblast ⇒ responsible for nutrition

Ectoderm
Mesoderm
Entoderm
Trophoblast
Amniotic sac
Yolk sac

Two small cavities appear, one in the ectoderm forming the amniotic sac, the other in the entoderm - the yolk sac.

Projections of proliferating trophoblast cells (primitive villi) invade decidual vessels
Mesodermal stalk
Embryonic area
Mesoderm
Chorion
Chorion

Amniotic sac
Embryonic disc
Mesoderm
Yolk sac
Trophoblast
Connecting stalk
Mesoderm
Amnion
Amniotic sac
Embryonic disc



Implantation:

- It occurs on the 6th to 7th days after ovulation
  ↘ by penetration of the trophoblast into the DECIDUA ¤ :-
  ↘ modified secretory endomet. under effect of both **P✓** & **E**

- Functions of the decidua ¤
  1- Site of........................... *implantation & nutrition of blastocyst*
  2- Site of.......................................*formation of the placenta*
  3- Protection.................*against laws of transplantation immunology* → Protect baby from mother's immune system
  4- Protection...................*against the invasive power of trophoblast*
  ↘ invasion stops at a fibrinoid layer called **Nitabüch**
  ↘ absence of this layer → adherent placenta → failed pl. delivery (pl. accreta)

Formation of the chorionic villi

- The chorionic villus is formed of 2 layers of trophoblast :-
  - Cytotrophoblast = Langhan layer......inner
  - Syncitiotrophoblast.........................outer (loss of cell walls → Rapid movement of nutrients and waste products.)

- Chorionic villi are 2 types

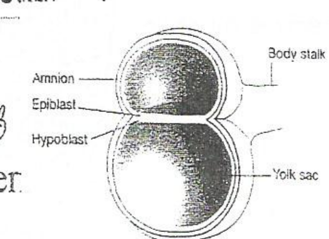
◗ Anchoring ➠ ❶ Fixation & attachment
.*Solid* finger like projections into the decidua → **1ry villi**
.*Mesoderm* develops in the 1ry villi (CT core) → **2ry villi**
.*Vascularization* of 2ry villi with fetal vessels → **3ry villi**

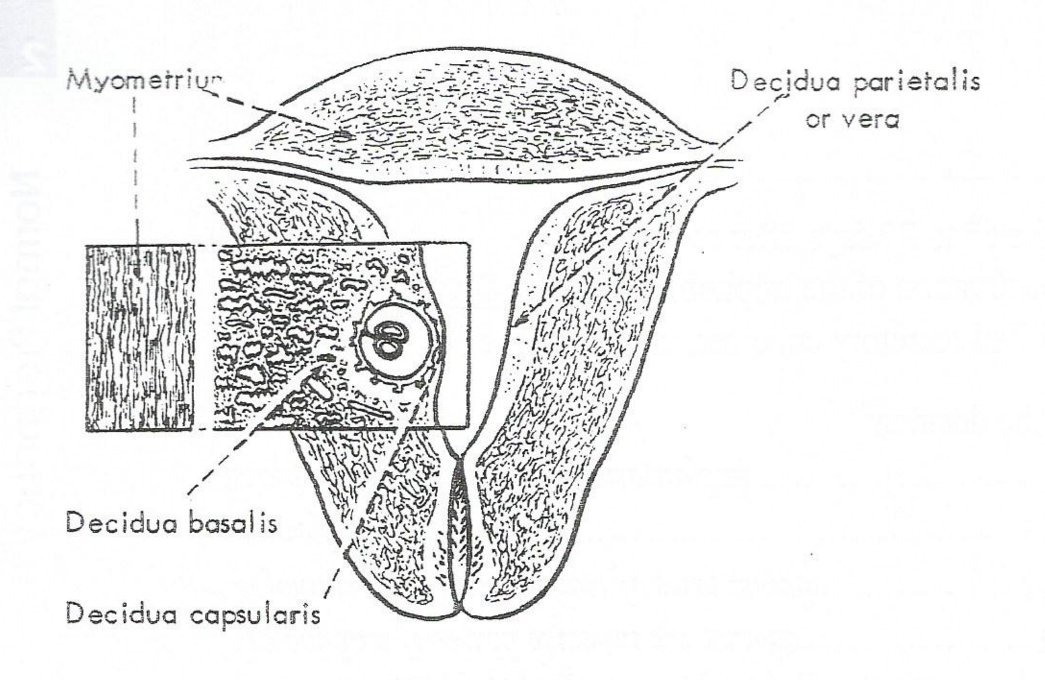
◗ Free villi (floating. Branches of Anchoring villi.) ➠ ❷ Food & nutrition
.The trophoblast invades the endometrial vessels (spiral a.) → form chorio-decidual space).
→ **1ry wave of invasion**
.At **20 weeks** trophoblast invades *media* of spiral vessels → **2ry wave of invasion** (Converts it into bl. sinusoids)

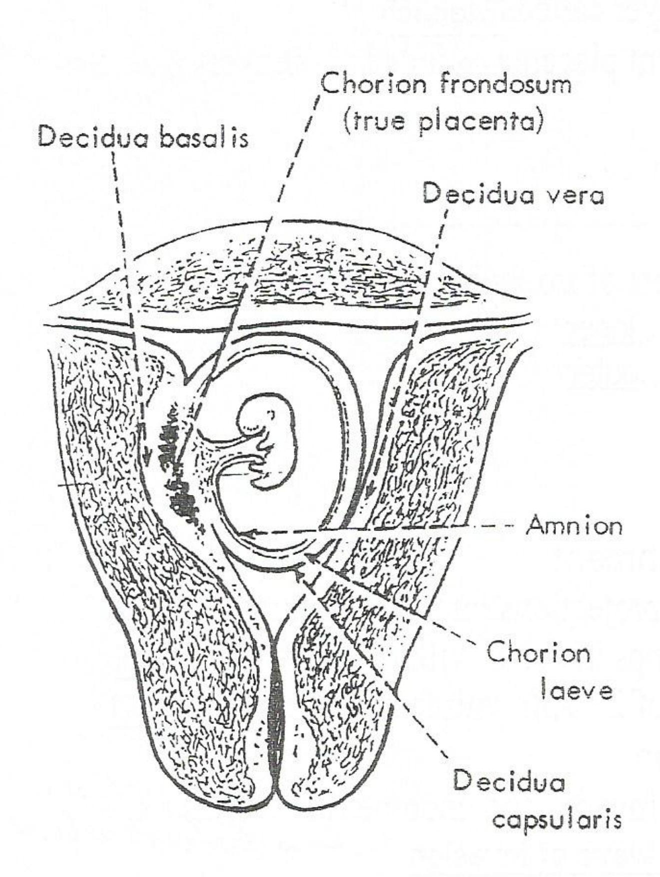
➠ ❸ Functional unit ¤ (Endocrinal funct.).
.HCG → maintenance of CL to produce 'P' for 7–10 wks till the placenta is formed

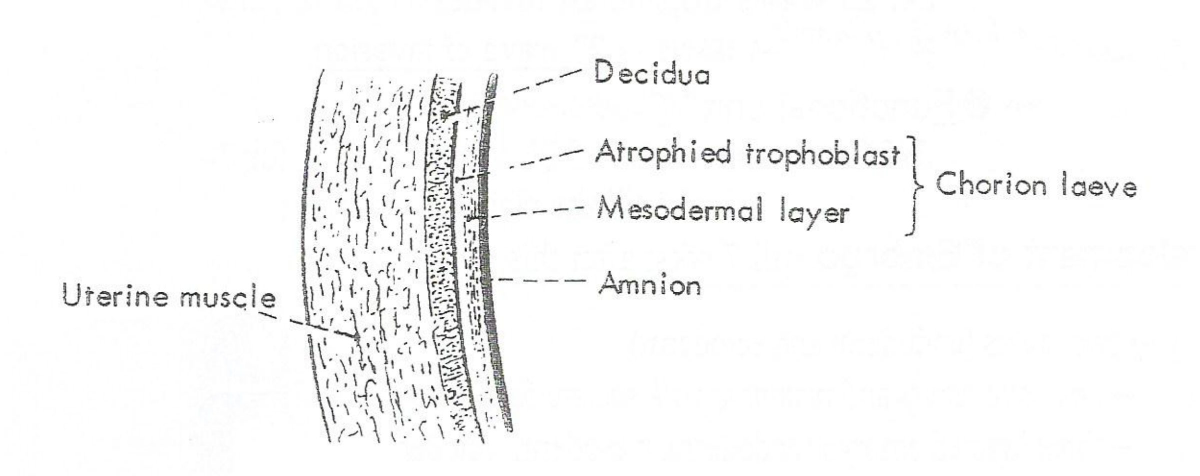
Early development of Embryo (till 7 wks; after this → a fetus ¤)

- 7th day → two layers (endoderm and ectoderm)
- 10th day → *amniotic cavity* and *primitive yolk sac* are formed ✌
- 16th day → three layered embryo (endoderm, mesoderm, ectoder




Myometrium
Decidua parietalis
or vera
Decidua basalis
Decidua capsularis


Chorion frondosum
(true placenta)
Decidua basalis
Decidua vera
Amnion
Chorion
laeve
Decidua
capsularis


Decidua
Atrophied trophoblast
Mesodermal layer
Chorion laeve
Amnion
Uterine muscle

Placenta

Normal structure

- **Shape**................discoid
- **Weight**..............500 gm ¤
- **Site**...................UUS (60% posterior) → site of implantation
- **Size**..................18–20 cm in diameter
- **Thickness**..........2.5 cm in center → gradually thins towards periphery
- **Cord insertion**......eccentric ¤

Placental formation

- **Two surfaces**
  - ❶ FETAL SURFACE is smooth & covered by amnion ¤
  - ❷ MATERNAL SURFACE is divided into 15-20 cotyledons (lobes)

- **Two parts** ¤
  - ❶ FETAL PART ⇨ **Chorion frondosum** (chorionic plate) = Trophoblast + mesoderm + fetal vessels projecting as villi into the intervillous space

    Chorion leave
    ↳ is the rest of chorion not sharing in placental formation

  - ❷ MATERNAL PART ⇨ **Decidua basalis** (decidual / basal plate)

    Decidua capsularis
    ↳ is the rest of the decidua overlying the developing ovum

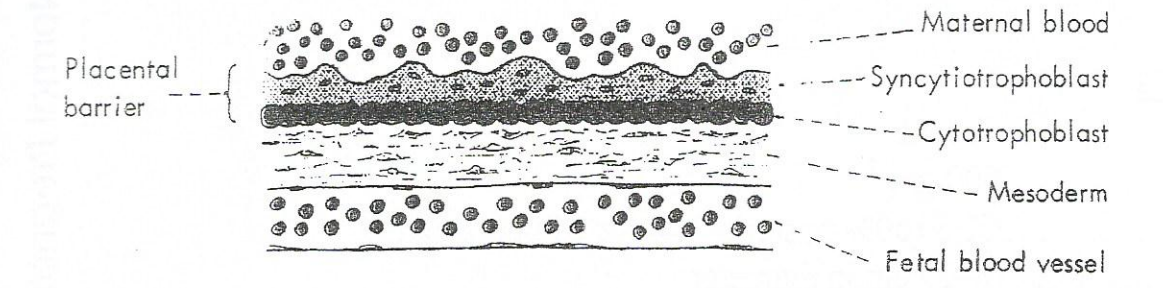
    Decidua parietalis (vera)
    ↳ is the decidua covering the rest of the uterine cavity

-Before 12 weeks → uterus is a pelvic organ.
-After 12 w. → uterus is a pelvi-abdominal organ

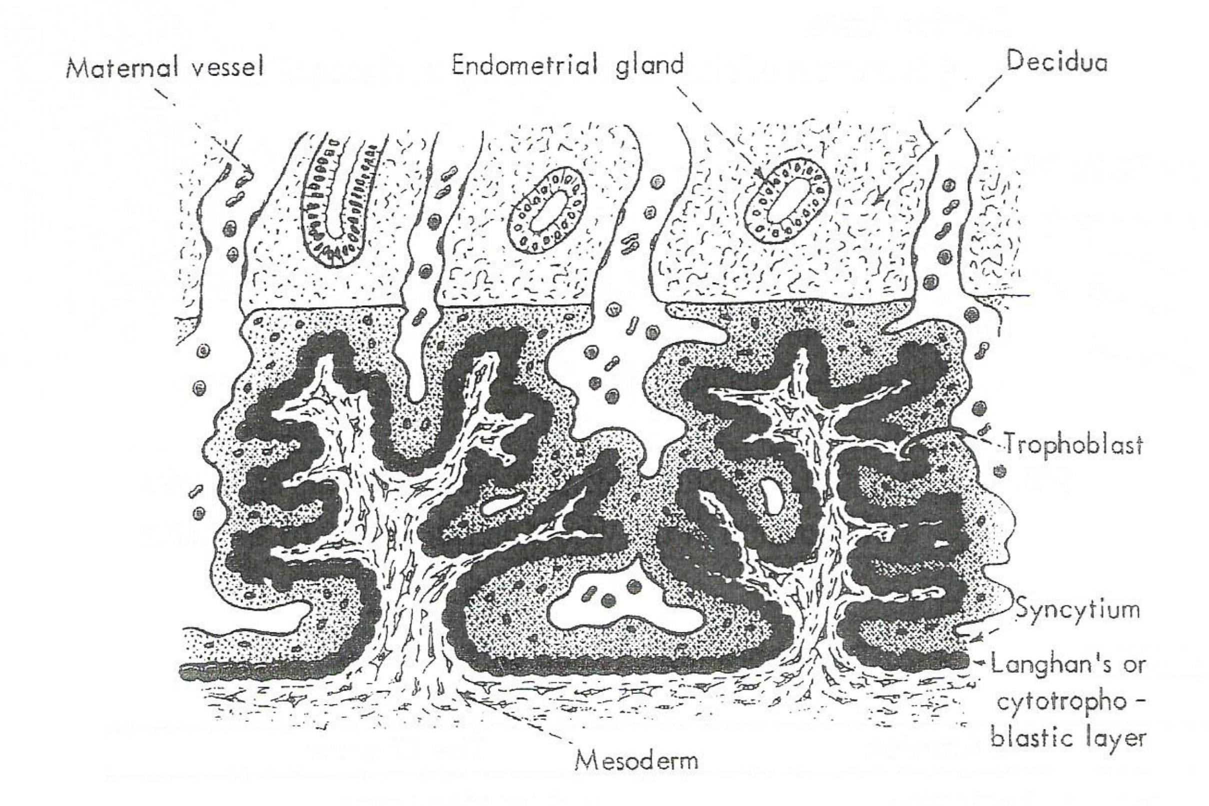
NB ⇨ Fusion of D. capsularis & D. parietalis occurs at **12** wks
↘ obliteration of the whole uterine cavity by the fetus

Fetal membranes.....................**2 membranes (could be separated)**

The Amnion	The Chorion
- The inner membrane	- The outer membrane
- Covers the fetal surface of the placenta & the cord	- In contact with the uterine wall, it ends at the margin of the placenta
- Transparent, glistening	- Less transparent



Maternal blood
Placental barrier
Syncytiotrophoblast
Cytotrophoblast
Mesoderm
Fetal blood vessel



Maternal vessel
Endometrial gland
Decidua
Trophoblast
Syncytium
Langhan's or cytotropho - blastic layer
Mesoderm

Functions of Placenta ΦΦ

❶ ▲ **Mechanical attachment**

▲ **Placental barrier:-** ¤

1- Cytotrophoblast 2- Syncitiotrophoblast (↓ at 5th – 6th month)
3- Mesoderm 4- Fetal capillary endoth. + its basement memb.

*The placenta becomes thinner as pregnancy advances ¤
*The placental is **permeable** to many drugs & organisms e.g.
- *Drugs* ☞ oral anticoagulants & oral hypoglycemics
- *Bacteria* ☞ TB, syphilis, malaria, toxoplasma
- *Viruses* ☞ MMR, CMV, chickenpox, polio

❷ ▸ **Respiration** (gas transport) by....................simple diffusion

▸ **Nutrition** ¤ **& excretion**
– Water & electrolytes...........simple diffusion
– Glucose, amino acids..........facilitated diffusion
– Ca, Fe, minerals..................active transport
– Immunoglobulins & LDL.......pinocytosis

❸ ▾ **Enzyme production** ⇨ oxytocinase.....alkaline phosphatase....insulinase

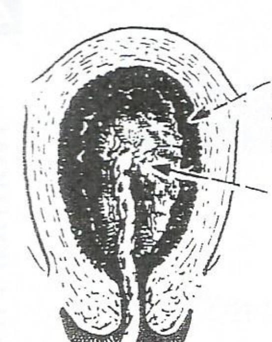
▾ **Hormone production** ⇨ sex steroids (estrogen & progesterone)

Source ⇨ < 7 weeks → from CL mainly
7-10 weeks → from CL+ placenta (syncitium ¤)
> 10 weeks → from placenta mainly
Function ⇨ responsible for all changes in preg
Level ⇨ they continue to rise till end of preg

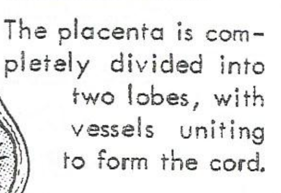
Feto-placental unit:
- Placenta can convert cholesterol (C_{27}) into progesterone (C_{21})
- Placenta can convert androgens (C_{19}) into estrogens (C_{18})
- However, placenta can't convert progesterone into androgens ∴ it must be supplied with androgens first e.g.:- DHEA & DHEA-S from both *maternal* & *fetal* sources (suprarenal gland mainly)
∴ ↓ in anencephaly ¤

Estrogen Level
- Estrone (E_1), Estradiol (E_2) → ⇧ 100 times
- *Estriol* (E_3) → ⇧ 1.000 times (the *index* ¤ ¤ of feto-maternal unit)
- Estetrol (E_4) → only formed in preg (of little significance)

Progesterone Level ➡ 50-150 ug/ml (10 times luteal level)

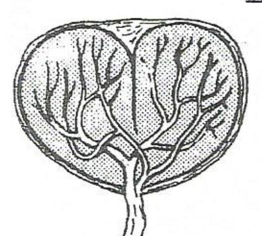


Placenta Membranacea



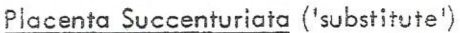
Placenta Duplex

The placenta is completely divided into two lobes, with vessels uniting to form the cord.

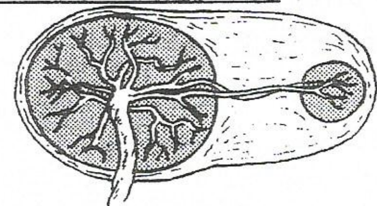


Placenta Bipartita

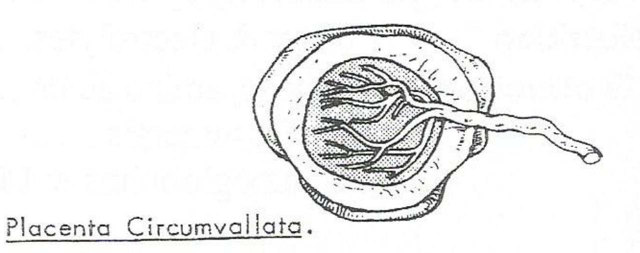
The placenta is partly divided into two lobes, with connecting vessels.



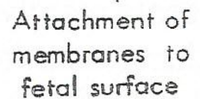
Placenta Succenturiata ('substitute')

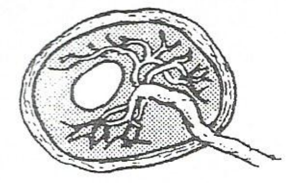


(A variant of Duplex)



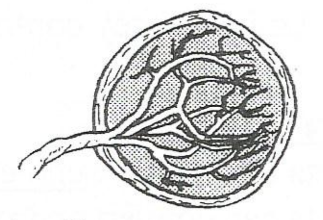
Placenta Circumvallata.





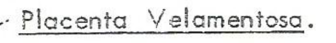
Placenta Fenestrata

A defective area appears in the middle of the placenta. It may be wrongly taken for the site of a missing lobe.

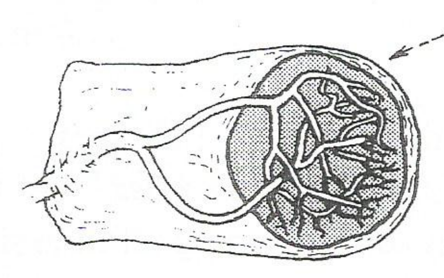


Battledore Placenta

Sometimes the cord has a marginal instead of a central insertion. This has no clinical significance.



Placenta Velamentosa.



Abnormalities of Placenta ✍ Φ

➤ Shape

1. **Bipartite placenta** ⇨ 2 equal lobes connected by……membranes
2. **Bilobate placenta** ⇨ 2 equal lobes connected by…… placental tissue
3. **Placenta fenestrata** ⇨ a window is present (a part of placenta is missed)

4. **Placenta succenturiata** (succenturiate lobe / lobes)
   - Small accessory cotyledon/s attached to placenta by membranes ¤
   - May be torn away during delivery → retention → PPHge ¤ or p.sepsis
   - Diagnosed by routine examination of pl. → site of torn vessels on margin

5. **Placenta circumvallate** (extrachorial placentation)
   - The chorionic plate (ch.frondosum) is < the basal plate (D.basalis)
   - The fetal margin shows a white ring formed of decidua
   - May lead to abortion…….CFMF, IUGR, PTL, IUFD……accidental hge

6. **Placenta membranacea**
   - The chorion leave does not atrophy → large thin placenta (15–20 inches)
   - May lead to placenta previa
   - If accompanied by vasa previa → APHge of fetal origin ✓

➤ Site

- In LUS → placenta previa
- On septum → liability to abortion, APHge, PPHge or retention
- Elsewhere (as tubes or peritoneum) → ectopic pregnancy

➤ Size

- Small (associated with IUGR or infarcts) ⇨ placental insufficiency
- Large (hyperplacentosis) ⇨ syphilis, Rh, DM, twins, placenta membranacea
  - . *Syphilis*: large, pale, friable / Endarteritis obliterans / Spirochetes
  - . *Rh isoimmunization*: large, pale, edematous

➤ Abnormal adherence (absent Nitabuch layer)

- P. accreta → placenta reaches basal layers of decidua, may reach muscle
- P. increta → penetrates muscle but does not reach serosal surface
- P. percreta → perforates uterus

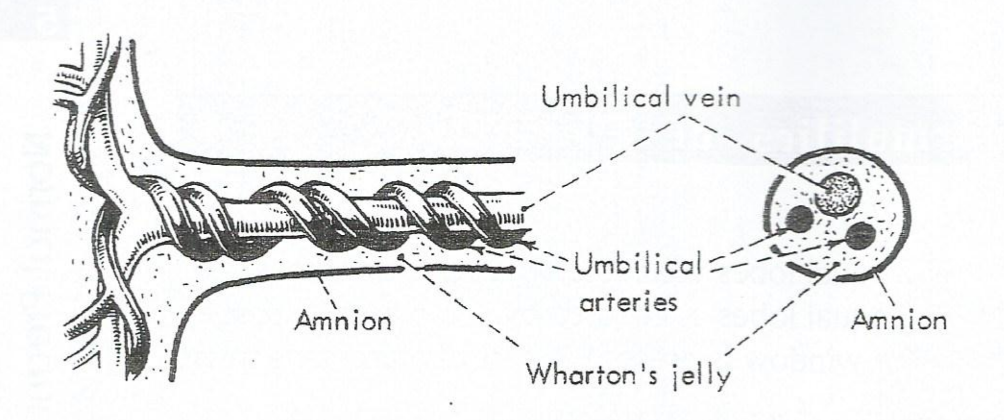
➤ Placenta infarctions → esp. in PET due to narrow blood v. → **red infarcts**

Normal calcification (physiological) → **white infarcts**

➤ Tumors of placenta

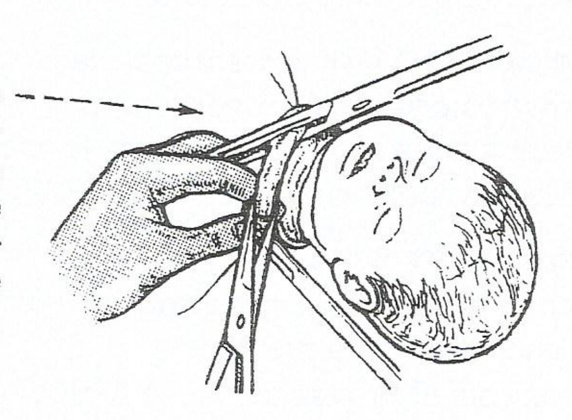
- Placental polyp → retained parts of placenta after delivery
- Vesicular mole & Choriocarcinoma
- Chorioangioma → vascular malformation (tumor) → polyhydramnios

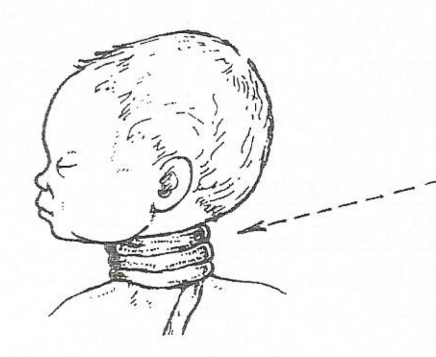
➤ Abnormal attachment of the cord…………



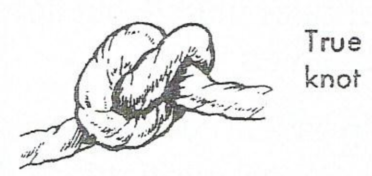
Cord Round the Neck

One or two loops of cord are quite often seen round the baby's neck at vertex delivery and normally do no harm. As soon as the neck is visible at the vulva the loop should be clamped and divided before delivery of the shoulders and trunk.



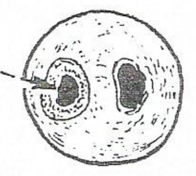


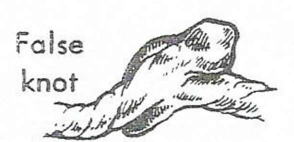
Much less frequently six or seven loops are drawn tightly round the neck. As the fetus descends the cord tightens, the blood supply is interrupted and the baby is stillborn. This is one cause of sudden acute fetal distress.



Single Umbilical Artery

This abnormality is frequently associated with other congenital abnormalities in the fetus.





Umbilical Cord [funis]

Structure

- Length → about 50 cm
- Diameter → about 2 cm
- Contents → 2 arteries (non-O_2) & 1 vein (O_2) ¤ carrying *fetal blood* along with remnants of allantois in myxomatous tissue (Wharton's jell). Vessels are convoluted (length of vessels > cord)
- The amniotic membrane covers the umbilical cord ¤

One vein carries **oxygenated** blood to the fetus
Two arteries carry **reduced** blood from the fetus to placenta

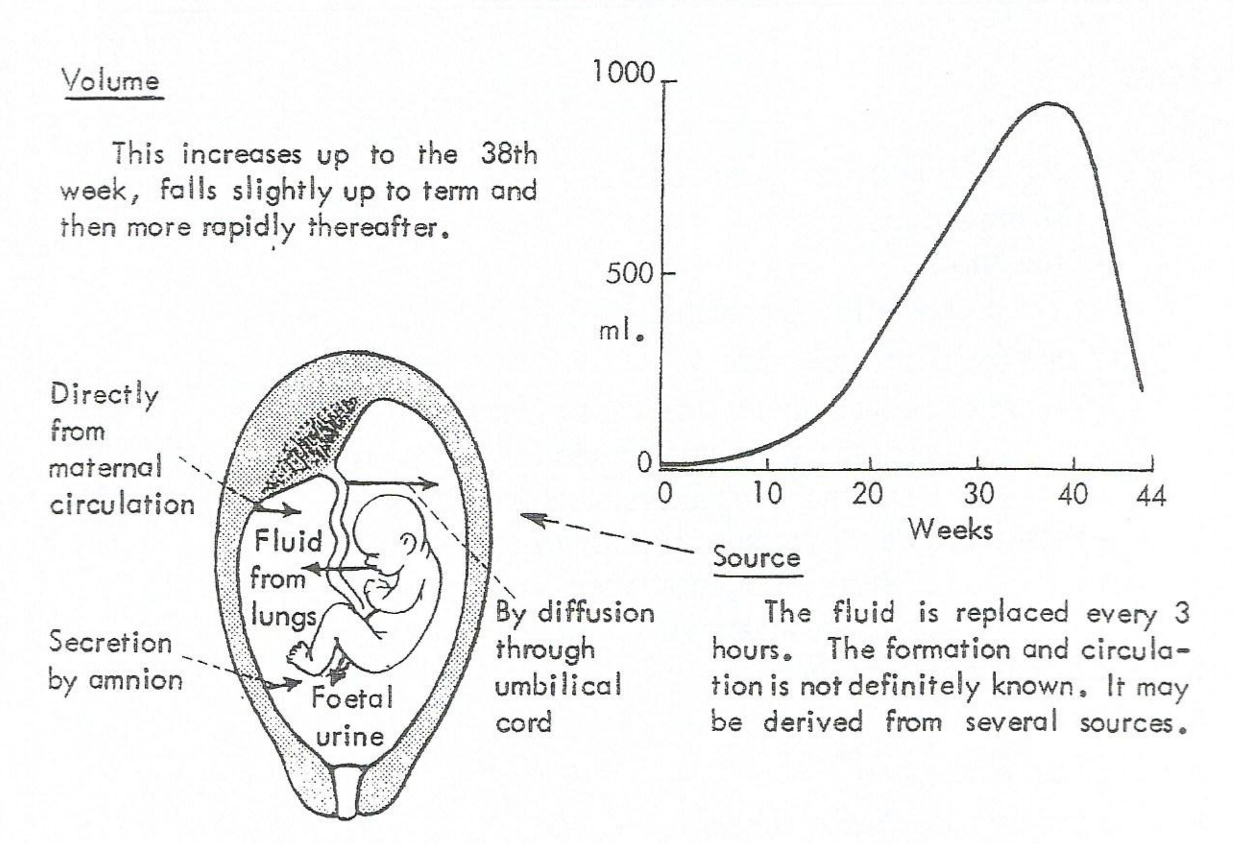
Abnormalities

1. Length
   - *Very long* (>100 cm) *may lead to:*
     - Coiling around fetus
     - True knots
     - Cord presentation & prolapse ¤
   - *Short cord* (<32 cm) *may lead to:*
     - Failure of . engagement & descent of fetus
       . presentation (malpresentation e.g. transverse lie)
       . external cephalic version or forceps
     - Fetal asphyxia (distress) or rupture of cord or
       . APHge (accidental hge)
       . Uterine inversion
2. Abnormal attachment: may be:
   - *Central*
   - *Marginal (battledore)* ¤
   - *Velamentous insertion of the cord*
     - Vessels are inserted into the membranes (& not placenta)
     - If the traversing vessels pass below the presenting part in the region of the cervix they are ⇨ called.....vasa previa
     - It is usually associated with placenta membranacea
3. Knots in cord, may be:
   * *True* ⇨ fetus passes through loops of the cord → may lead to fetal distress
   * *False* ⇨ localized varicosity in a collection of Wharton's jelly → no effect
4. Congenital umbilical hernia
5. Tumors / cysts (as myxoma and sarcoma)
6. Absence of one umbilical artery
   * Common in DM
   * May be associated with CFMF, IUGR, prematurity

AMNIOTIC FLUID

Volume

This increases up to the 38th week, falls slightly up to term and then more rapidly thereafter.



Source

The fluid is replaced every 3 hours. The formation and circulation is not definitely known. It may be derived from several sources.

Amniotic fluid

▶ **Layers of amnion** (0.5 mm → 5 layers)

1. Cuboidal epithelium
2. Basement membrane
3. Compact layer (reticular fibers arranged in bundles)
4. Fibroplastic layer
5. Spongy layer (contains mucous → can glide upon chorion)

▶ **Source of amnion**...........amniogenic cells (from fetal ectoderm)

▶ **Source of amniotic fluid**

1. Maternal ⇨ transudation (**esp 1st trimester**) through placenta & cord
2. Fetal ⇨ - Urine ✓✓ (**esp in the 2nd half**)
   - Lungs (bronchial, buccal & salivary glands)
   - Amniotic epithelium
   - Transudation from fetal skin & umbilical cord

↘ **Then it is removed** by transudation + fetal swallowing i.e. **dynamic circulation** ¤

▶ **Volume**

6 wk → 5 ml	**10 wk** → 30 ml	**20 wk** → 300 ml
30 wk → 600 ml	**36 wk** → 1.000 ml	**38-40 wk** → 800 ml

▶ **Composition**

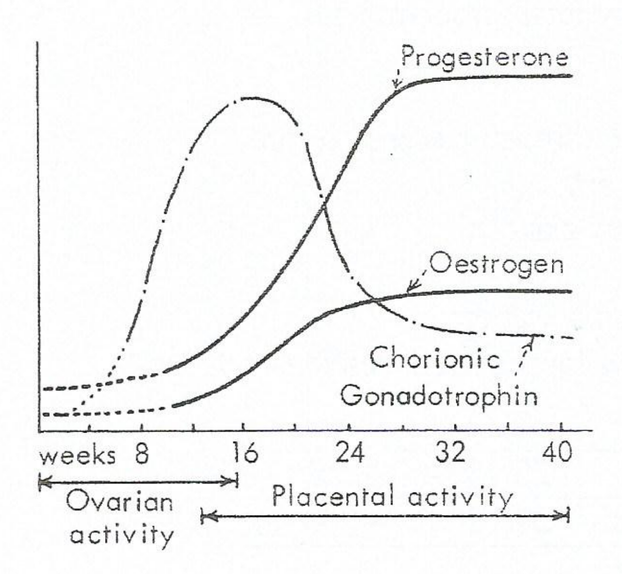
- **99 % clear watery**
- **1–2% crystalloids & colloids** [+ vernix caseosa, desquamated epith., lanugo hair]
  - CHO (glucose & fructose) – proteins (albumin & globulin) – lipids
  - Hormones (E.& Pr.) – electrolytes (Na. K. cl. Cu.)
- **Physical properties**
  - Colorless → later on will be turbid
  - Specific gravity → 1010-1020
  - Reaction → slightly alkaline ¤ (7-7.5)

▶ **Functions** Φ

1. During pregnancy		2. During labor
.Protection against trauma	. Thermoregulation	. Helps cervical dilatation
.Prevents adhesions bet. fetal skin & amnion	. Allow free movement of fetus → muscular development	. Prevents direct fetal & placental compression by the uterine wall
.Prevents infection	. Development of alveoli	. Wash birth canal after ROM

▶ **Abnormalities**

- **Volume** (⇧...polyhydramnios, ⇩....oligohydramnios, PROM)
- **Meconium staining** ⇨ meconium aspiration syndrome
- **Inflammation** ⇨ chorioamnionitis
- **Amniotic cysts........bands** (may lead to amputations)



Roles of selected placental hormones

Hormone	Role
Human chorionic gonadotrophin (hCG)	Initially maintains the corpus luteum's secretion of progesterone and oestrogen; later it may have a role in regulating placental oestrogen secretion and in modulating the maternal immune response
Oestrogen	Over 90% is in the form of oestriol; it is involved in uterine growth, cervical changes, and breast development
Progesterone	Smooth muscle relaxation, acting on the uterus, gastrointestinal tract and ureters. Also has a role in regulating maternal physiological changes
Human placental lactogen (hPL)	Mobilizes maternal free fatty acids, improving glucose availability for the fetus

Hormones

✵ **Steroids** ➟ estrogen & progest...........from CL & placenta ¤

✵ **Proteins** ➟ HCG & HPL...............from syncitio-trophoblast ¤

1) Human chorionic gonadotrophin

▸ ***Time of production*** (a glycoprotein) ¤
- Appears at 1st day of implantation
- Can be detected within 10 days of fertilization (conception) ¤
  ↘ i.e. before missing a period

▸ ***Level***
- It ⇧ rapidly in early pregnancy → level doubles every 2 **days**
- Reaches a peak at 70 day (**10 wk**) gestation (≈ **50.000 mIU/ml**) ¤
- Then it ⇩ at 100 d (**14 wk**) ≈ (**5.000**) & remains as such till term
  ↳ formation of Pl. → produces P + E → No need for CL.

▸ ***Disappears at***
. 1–2 weeks after abortion
. 2–8 weeks after labor
. 8–12 weeks after vesicular mole evacuation

▸ ***Assessed by***

Urine → **Slide agglutination with latex** (detects 500 mIU/ml)

شرايط البول بيكتشف الحمل أول أسبوع بعد غياب الدورة ← قبل افتقاد الدورة

ELISA: more sensitive (90%), (detects 50 mIU/ml)

Serum → **RIA** ✓✓ the most sensitive, (detects 5 mIU/ml)
↘ assess β-*subunit* (as α-*subunit* is similar to FSH, LH, TSH)
↳ 145 Amino acid → 92 A.A.

▸ ***Value***

Function	Uses
Maintenance of CL (luteotropic ¤)	. Diagnosis of pregnancy
Immunological suppressive action	. Diagnosis of pregnancy abnormalitie
Stimulates fetal testosterone sec. ¤	. Diagnosis and follow up of V. mole

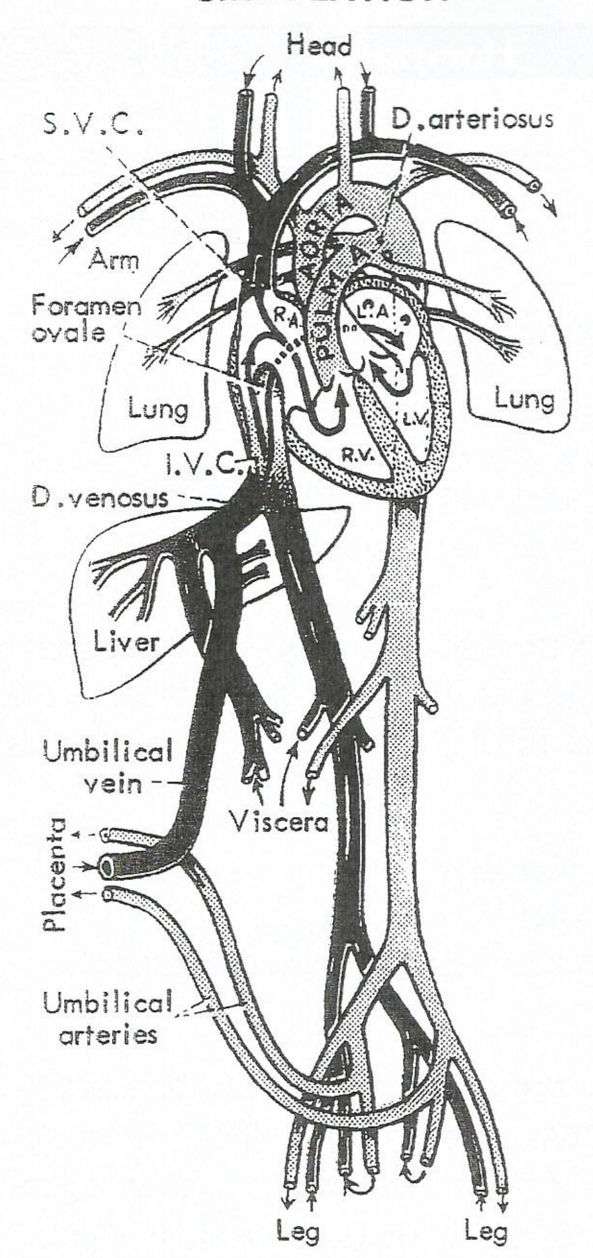
2) Human Placental Lactogen (a polypeptide) ¤

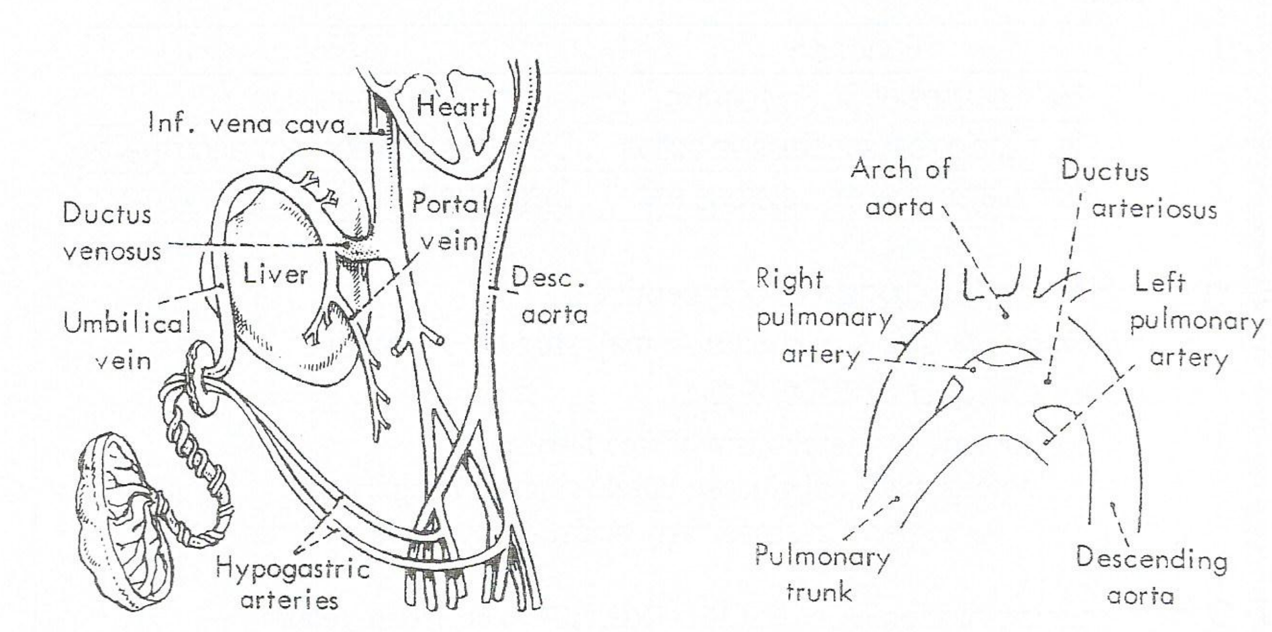
▸ ***Very similar to GH & prolactin*** → may stimulate growth of breasts

▸ ***Anti insulin effect on CHO & fat*** ¤
. Lipolytic → metabolism of free fatty acids
. Inhibits maternal glucose uptake & gluconeogenesis
↘ spares glucose, fatty acids, amino acids for fetus

3) Others
- human chorionic GnRH, CRH, TRH, ACTH, prolactin, **relaxin**
- **Placental activin** → stimulate GnRH & HCG while **inhibin** → inhibit them

CIRCULATION





Fetal circulation

❶ Intrauterine

- Oxygenated blood from the placenta passes to the fetus via the **umbilical vein (1)** ¤ → penetrates liver to give it small branches
- Most of the blood is directed via the **ductus venosus** into the IVC (which carries also the returning non-O_2 blood from LL ¤)
- There is only partial mixing of the 2 streams and most of the oxygenated blood is directed by the **crista dividens** (at the upper end of the IVC) through the **foramen ovale** into the left atrium → the left ventricle → aorta → this relatively well O_2 blood supplies → the head & UL
- The remainder of the blood from the SVC mixes with that of IVC → passes to the right ventricle → very small amount of blood goes to the lungs (high pulmonary vascular resistance ¤). Most blood passes via the **ductus arteriosus** to the aorta (beyond the vessels supplying the head & upper extremities) → supply viscera & LL
- Little blood actually goes to the LL. Most of it passes into → Rt & lt internal iliac arteries → **umbilical arteries (2)** ¤ : non O_2 blood

To compensate for the low arterial PO_2 in the fetus:- ¤
* Increased fetal cardiac output
* Increased fetal systemic blood flow rates
* Increased affinity for O_2 (↑ fetal O_2 carrying capacity = Hb-F)

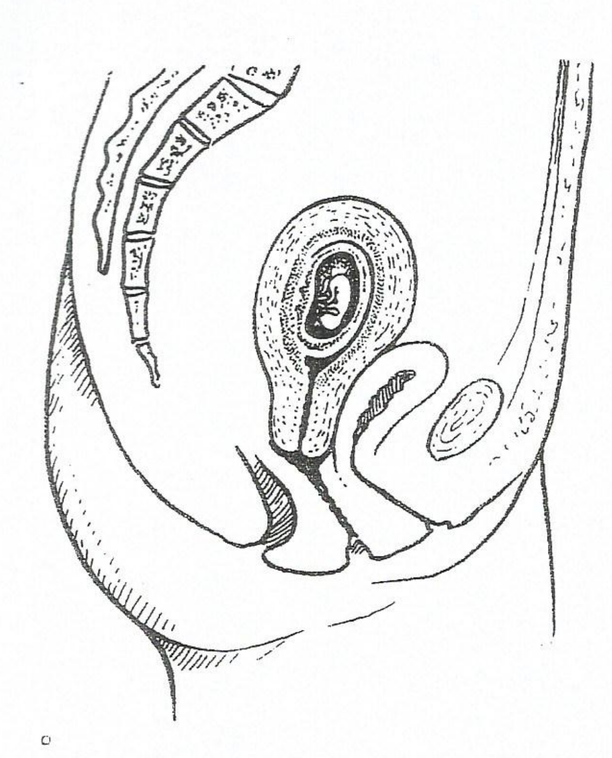
❷ At birth

- The umbilical vessels contract in response to ↓ temp → ↓ O_2 tension & ↑ CO_2 → stimulation of respiratory center
- Breathing → –ve thoracic pressure → sucks more blood from the pulmonary artery into lungs & diverting it from the ductus arteriosus which gradually closes
- The left atrial pressure → closes the foramen ovale

❸ Later

- Umbilical vein → **ligamentum teres** (runs in the free border of the falciform ligament in the adult)
- Umbilical arteries → **hypogastric ligaments** (lat. umbilical lig) ¤
- Ductus venosus → **ligamentum venosum** ¤
- Ductus arteriosus → **ligamentum arteriosum** ¤

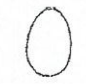
Maternal adaptation



7 weeks

PALPABLE UTERINE ENLARGEMENT

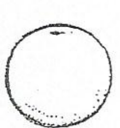
At 7 weeks the uterus is the size of a large hen's egg

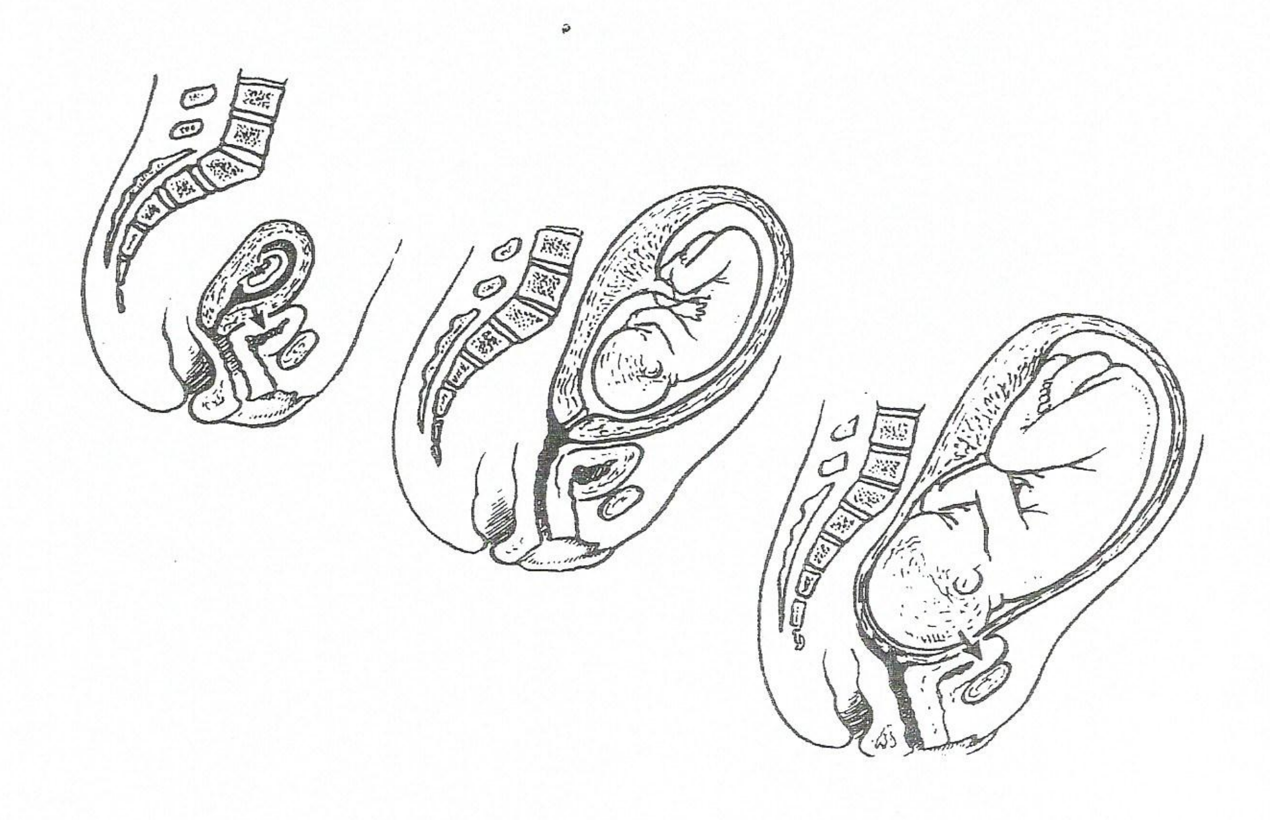


At 10 weeks it is the size of an orange



At 12 weeks it is the size of a grapefruit





Maternal adaptation

① The genital tract

1) The uterus

☆ **Shape** → ⇧ from **50 gm** (10 ml^3) to → **1 kg** (5000 ml^3)
→ changes from pear shape → globular → pyriform

☆ **Size** → 8 wks (5 cm).....12 wks (10 cm).......16 wks (15 cm)
→ Then fundal level according to gestational age
→ Till reaching 35–40 cm at term

☆ **Position** → *dextrorotated* (Lt round ligament becomes nearer to midline)
uterus must be centralized before CS.
Pyelonephritis is more common on Rt side.
Lt uterine wall is more stretched → more liable for rupture.
→ *dextroflexed* (d.t. presence of sigmoid colon)
→ *soft consistency* (d.t. vascularity → 500 ml/min ¤ – & amniotic fluid)

☆ **Myometrium**

- *Hypertrophy* ✓ & *hyperplasia* of muscle fibers ↑P,E
- *Contractility*
  - In early preg., they are detected bimanually ➡ Palmer's sign
  - Later on, cont. are detected abdominally ➡ Braxton Hick's
  - They become perceptible & painful near term ➡ false labor pain

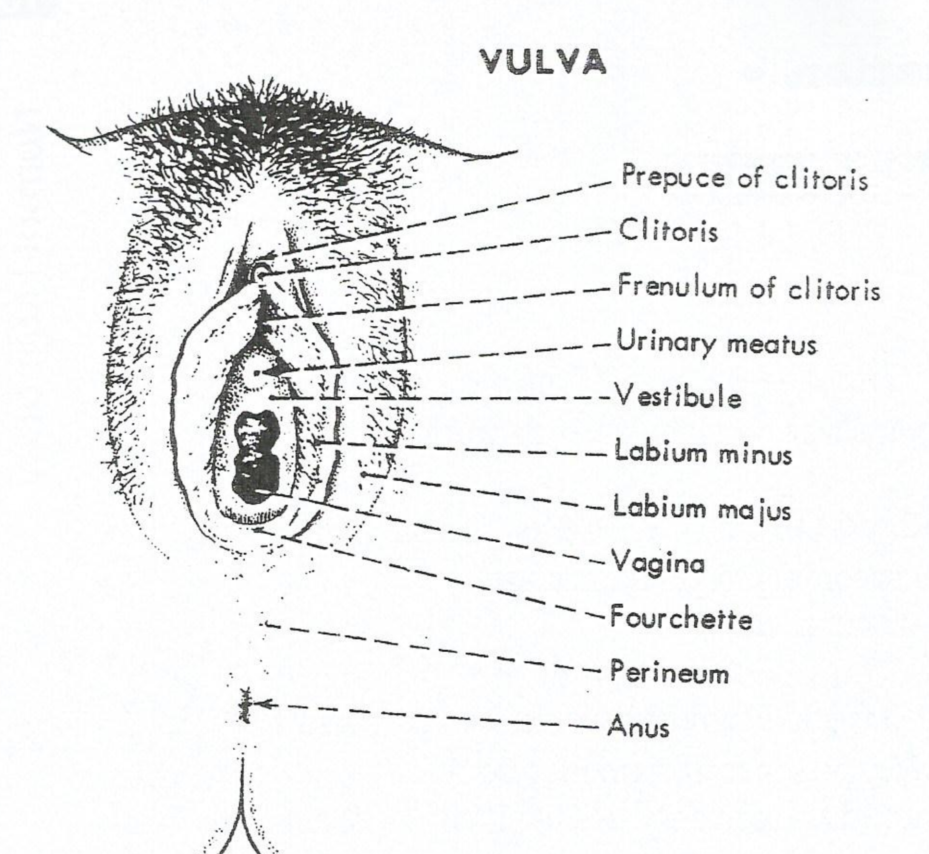
☆ **Formation of lower uterine segment**

- Formed from isthmus (bet. <u>A</u>natomical os above & <u>H</u>istological os below)
- It is 4 mm in length and is covered anteriorly by loose peritoneum.
- During pregnancy & labor → it is stretched to 10 cm
- It differs from the upper segment in

	Upper segment	Lower segment
Peritoneum	Adherent	Loose
Muscle	Thick (3 layers)	Thin (2 layers)
Decidua	Well developed	Less developed
Membranes	Firmly adherent	Loosely adherent
Action	Active in labor (contracts & retracts)	Passive ¤ (dilates & stretches)

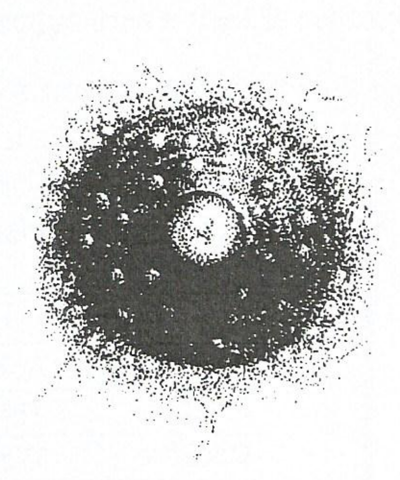
- <u>Physiological retraction ring</u>
  It is a groove between the thick UUS & thin LUS below the symphysis pubis. Normally it is not *seen* or *palpable*

VULVA





The breast at 8 weeks



The breast at 16 weeks

2) The ovaries

- **No ovulation occurs** (suppressed LH & FSH) → due to ↑P, E
- **The corpus luteum secretes**
  - **E & Pr** → produced mainly from CL till 7 wks, then production is shared with placenta till 10–12 wks, then CL will gradually ↓ in size
  - **Relaxin** → a protein hormone of unknown function. May have a role in ripening of cervix & relaxation of pelvis at labor
- **A CL cyst** may be found in the 1st trimester → may be misdiagnosed as Ectopic Preg. by U/S.
  ⊊ < 6 cm disappears spontaneously (functional)

o Preg. luteoma is solid swelling in ovary due to presence of excess theca cells in CL → ↑ Androgen sec. effect → mother → virilization (transient); Baby → virilizing of ♀ fetus → never occur as placenta contain aromatase enz.

3) The fallopian tubes ⇨ enlarged, stretched, increased vascularity

4) The vulva

- ⇧ ed vascularity ⇨ soft & violet (Jacque Meir sign)
- ⇧ ed Liability to ⇨ varicose veins & edema

5) The vagina

- ⇧ ed vascularity ⇨ soft & violet (Chadwick sign)
- ⇧ ed secretions ⇨ acidic (lactobacilli)
- Epithelium is thick (smear → intermediate cells)

6) The cervix

- ⇧ ed vascularity ⇨ soft & violet (Goodell's sign)
- ⇧ ed secretions ⇨ mucous plug obstructing cx canal (operculum)
- Epithelium:- ectopy (replacement of st.sq.epith of ectocx by columnar) → misdiagnosed as ulcer.

2 The breast

*** The 1st few weeks** ⇨ tenderness, tingling
(d.t. ↑size, vascularity & sensitivity of nipples & breast)

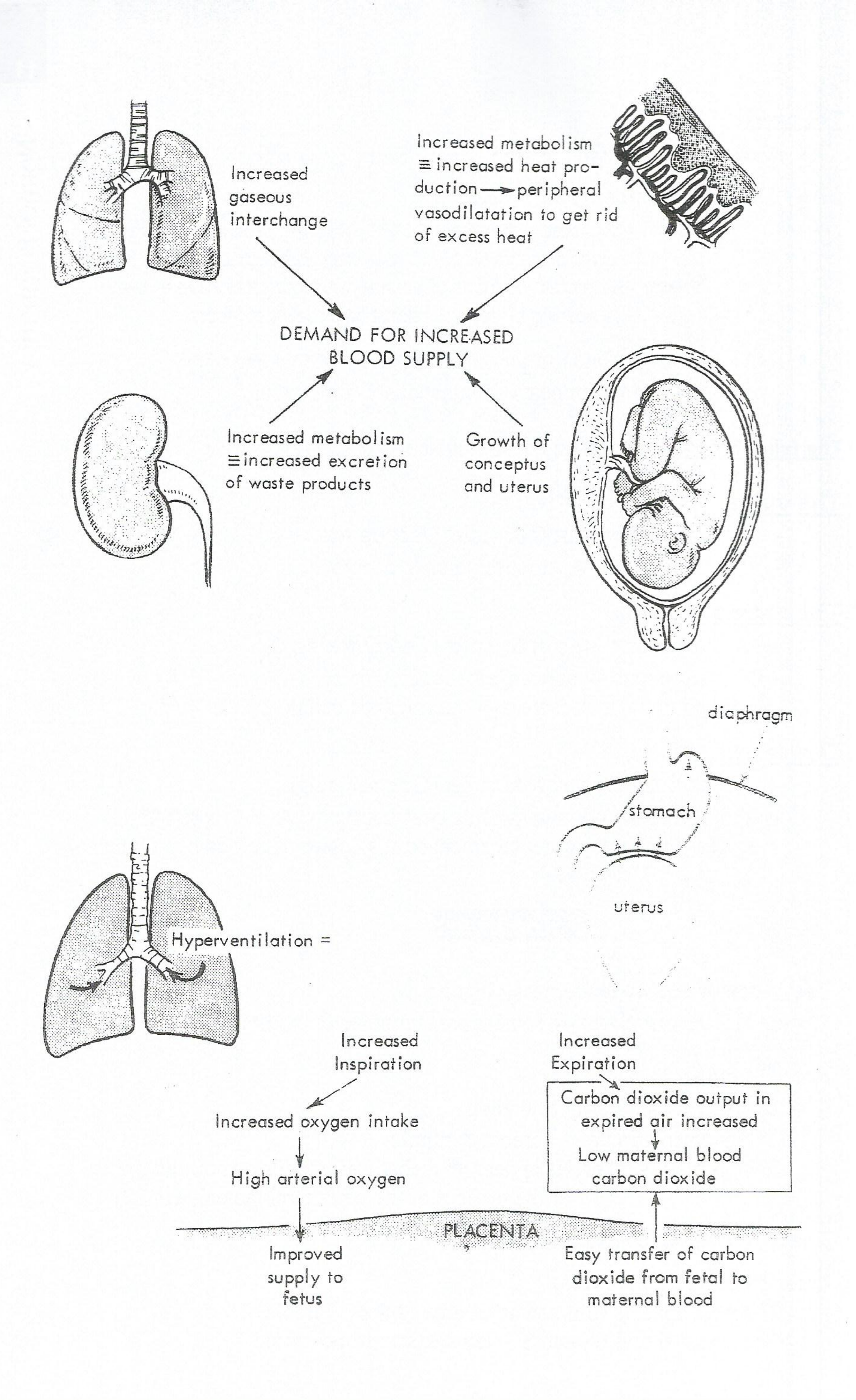
*** The 2nd month**

- ⇧ ed size & nodularity of breasts
- ⇧ ed pigmentation of 1ry areola & nipple → permanent.
- Montgomery's tubercles appear (sebaceous glands **or** pouting lips of the orifices of the peripheral lacteals 10–20)

*** The 3rd month** ⇨ colostrum appears

*** Later on (5th or 6th months)**

- 2ry areola appears (pigmentation around the 1ry areola)
- The breast shows dilated SC veins & sometimes striae


Increased
gaseous
interchange
Increased metabolism
≡ increased heat production → peripheral
vasodilatation to get rid
of excess heat
DEMAND FOR INCREASED
BLOOD SUPPLY
Increased metabolism
≡ increased excretion
of waste products
Growth of
conceptus
and uterus
diaphragm
stomach
uterus
Hyperventilation =
Increased
Inspiration
Increased
Expiration
Increased oxygen intake
High arterial oxygen
Carbon dioxide output in
expired air increased
Low maternal blood
carbon dioxide
PLACENTA
Improved
supply to
fetus
Easy transfer of carbon
dioxide from fetal to
maternal blood

Progesterone → Relaxant hormone of Preg.

③ The cardiovascular system Φ

► **The blood**

* **Pressure** ⟹ ⇩ esp in 2nd trimester

- Placenta acts as an AV shunt } leading to ⇩ in the P.resistance
- Vasodilator effect of progest. } & ⇧ of the peripheral flow

* **Volume of Plasma** ⟹ increase 40–50% (max at 30–34 weeks)¤

* **Elements**

- **RBCs** → increase 20–30% ¤

  So, there is more ↑ in plasma volume > RBC volume → physiological anemia (haemodilution). Pathological if < 11gm%

  → Haematocrit → decreases
- **Leukocytes** → ⇧ slightly, esp after labor (14–16.000 /ml^3)
- **Blood coagulation** → increased coagulability
  - ⇧^{ed} factors VII–X and fibrinogen + ⇩$_{ed}$ fibrinolytic activity
  - Platelets → mild decrease (haemodilution).
- **ESR** → increases (due to ↑ fibrinogen)

► **The heart**....changes occur from 1st trimester ¤

- ⇧^{ed} COP (30–50%) ⟹ (d.t. ⇧ both SV & HR = 10–15 bpm)
- The heart is displaced upward & laterally by the diaphragm

  → shift of apex beat from 5th to 4th intercostal space
- Due to increased flow rate
  - **Heart sounds**

    . Splitting of the 1st sound

    . Appearance of the 3rd sound/hyperdynamic circulation
  - **Heart murmurs**

    . Soft systolic murmur may be present (90%)

    . If diastolic murmur occur we must exclude *pathology*

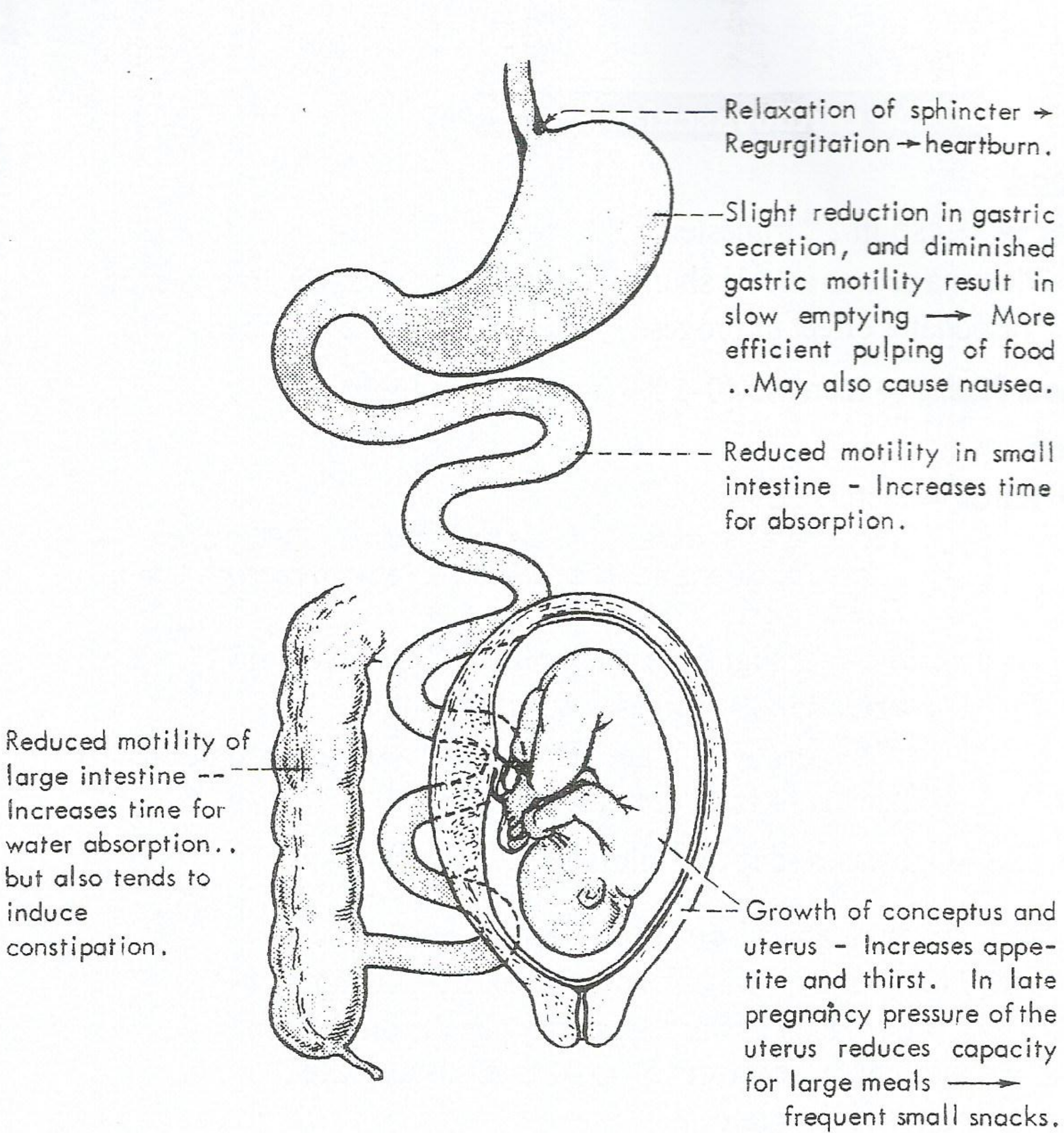
► **Veins** There is increased liability to varicose veins due to (more in Lt LL due to pressure of Rt common iliac a. on the Lt common iliac V.)

- Progesterone (relaxant effect on vessels)
- Pressure of the gravid uterus
- Increase in blood volume

④ The respiratory system

- ⇧ ed dyspnea in late pregnancy → pressure & hormonal effect (Progesteron → ↑R.C → hyperventilation.)
- ⇧ ed tidal volume → reserve volume decreases
- ⇧ ed minute respiratory volume } progesterone effect
- ⇧ ed minute oxygen uptake } resp. alkalosis ¤ → due to hyperventilation → CO_2 wash.

No change. What about the ¤ RR.Q



The ureters are said to dilate greatly and again urine may stagnate.

Relaxation of bladder may result in incomplete emptying and collection of residual urine.

Both of these changes tend to favour the onset of urinary infection.

Hyperemesis gravidarum: vomiting ... degree that affect general condition (known by ketone bodies in urine).

لماذا تكره النساء الحمل؟

5 The gastrointestinal tract

Pr → Relaxation.

1) The mouth

- Morning sickness ⇨ nausea & sometimes vomiting in early pregnancy
- Changes in appetite ⇨ longing (pica) → desire to certain food
- Ptyalism ⇨ excessive salivation (hyperemia of the gums ± hypertrophy)

2) Esophagus

- Relaxation of cardiac sphincter } leads to pyrosis
- Delayed gastric emptying } = heart-burn (P. effect)

3) Stomach & intestine

- Delayed emptying, decreased motility
- ↓ acidity of stomach (hypochlorohydria) → regurgitation of alkaline chyle

4) Liability to constipation & piles ⇨ d.t. effect of Pr & pressure of uterus

5) The liver & gall bladder

- Changes in some liver enzymes esp alkaline phosphatase
- Tendency to cholestasis (↑Pr) → ↑Bile salts → itching.
- ⇧^{ed} globulin + ⇩$_{ed}$ albumin

↳ ↑ all binding globulins + ↑ IGs.

6 The urinary tract

► **The kidney**

- Increased size (by 1-1.5 cm)
- Increased renal blood flow → increased GFR (50%)
  - ↑ creat. clearance → ↓ serum creatinine (0.5 mg%), uric (3 mg%), BUN (8.5mg%)
  - ↓ renal threshold to glucose, amino acids & H_2O soluble vitamins

► **The ureters**

- Enlarged, dilated & tortuous (d.t. progesterone & pressure at pelvic brim)
- More on right side (d.t. dextroflexion of uterus)
- Increased liability to pyelonephritis

► **The bladder**

- Hyperemia
- ↑ frequency of micturition (1st trimester) & (last month d.t. engagement)
- Displacement upwards (SUI may be normal in late in pregnancy)

↳ Urine Incompetence.

7 The nerve & muscle

- Mood changes (elevated or depressed) – (sleepy or insomnia)
- Relaxation of the pelvic joints, sometimes arthropathies (Relaxin h).
- Lumbar Lordosis → to compensate for the enlarged uterus



CARBOHYDRATE METABOLISM

Increased intake of carbohydrate

More sugar to blood

Blood glucose

To tissues for oxidation

Insulin

Less glycogen in liver

Depot fat

Insulin

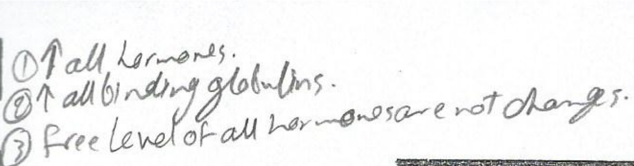
Insulin antagonists
Growth hormone substance
Placental corticoids

Placenta

Energy
Glycogen
Fat

Less muscle glycogen

Slight spill of sugar in urine



8 The endocrine glands

- **The pituitary**
  Increased size & vascularity (esp anterior lobe: 2-3 folds)
  ↑ prolactin – ↓ FSH & LH, GH [other hormones are unaffected]

- **The thyroid**
  Slight enlargement (↑ TSH & chorionic thyrotropin)
  <u>↑ serum thyroxine</u> ¤ (due to ↑ TBG, free hormones are <u>unchanged</u>)
  ↑ BMR 25% (due to pregnancy & not hyperthyroidism) ↳ Hyperdynamicity

- **The parathyroid**
  Slight enlargement → ↑ PARATHORMONE to ↑ serum Ca → Provide Ca for fetus.
  but CALCITONIN also ↑ ∴ <u>no change</u> in ionized Ca level

- **The suprarenal gland**
  Little morphological changes
  ↑ aldosterone & renin (due to ↑ CBG (→ Cortisone binding globulin), free serum cortisol ¤ are <u>unchanged</u>)

9 The skin and appendages

1. **Pigmentation**......d.t. ⇧ placental (→ chorionic MSH) & adrenal steroids, also E. may have MSH like activity
   - <u>Esp in</u> nipple, areola, axilla, vulva
   - <u>Linea nigra</u> (dark brown line between umbilicus & symphysis)
   - <u>Chloasma gravidarum</u> (butterfly pigmentation on face)

2. **Striae gravidarum**......due to ⇧ corticosteroids or by mechanical stretching
   - Pink lines due to rupture of elastic fibers or SC vessels of skin of abdomen (common), breast, thighs, buttocks. Later on after delivery → striae albicans

3. **Divarication of recti**
4. **Hyperemia** → vascularity of skin & m.m. (nasal congestion) + Common epistaxis.
5. **Sometimes** → falling of hair, palmar erythema and spider naevi

10 Metabolic changes

- **Body weight** ⇨ increases 12.5 kg on average (9-12.5 kg)
- **Salt & water retention** ⇨ due to increased aldosterone
- **Protein metabolism** ⇨ +ve nitrogen retention (1 kg increase during pregnancy)
- **CHO metabolism** ⇨ pregnancy is potentially diabetogenic
- **Lipid metabolism** ⇨ increased blood lipids & cholesterol ¤ → central fat deposition
- **Vitamins** ⇨ ⇧^{ed} fat soluble vitamins + ⇩$_{ed}$ water soluble vitamins
- **Minerals** ⇨ ↓ serum iron (Fe stores may be depleted if no Fe supplementation is given)
  But ↑ transferrin (total iron binding capacity) ↳ The only supplementation required in Preg.
- **Immunoglobulins** ⇨ increased levels of IgA and IgM

Minor Disturbances in pregnancy

❶ Gastrointestinal

- **Morning sickness....**
- **Gingivitis:** hyperemic gums that may bleed with the use of a tooth brush
- **Ptyalism:** excessive salivation
- **Heartburn:** Treated by antacids, more frequent meals, avoidance of spices
- **Indigestion:** hypochlorhydria (regurgitation of alkali chyle into stomach)
- **Constipation:** ⇧ fluid intake ± eating whole meal bread [& not white bread]
- **Hemorrhoids:** usually regress after delivery [but not completely]

❷ Urinary

- **Frequency of micturition:** d.t. pressure from the gravid uterus
- **Incontinence:** d.t. loss of the post urethra-vesical (PUV) angle

❸ Musculo-skeletal

- **Backache:**
  - Common in the last trimester
  - Treatment: . Avoid wearing high-heeled shoes
    . Exercises to strengthen the back muscles
- **Leg cramps:**
  - Electrolyte disturbance
  - Engorgement of lower limb veins
- **Round ligament pain:**
  - Sharp groin pains d.t. spasm of the ligament associated with sudden movements (esp the right side ---d.t. dextroposition)

❹ Skin changes

- **Striae gravidarum**
- **Sweating & feeling the heat:** (d.t. ⇧ peripheral circulation &VD)
- **Vaginal discharge = leucorrhea:** (d.t. ⇧ estrogen)

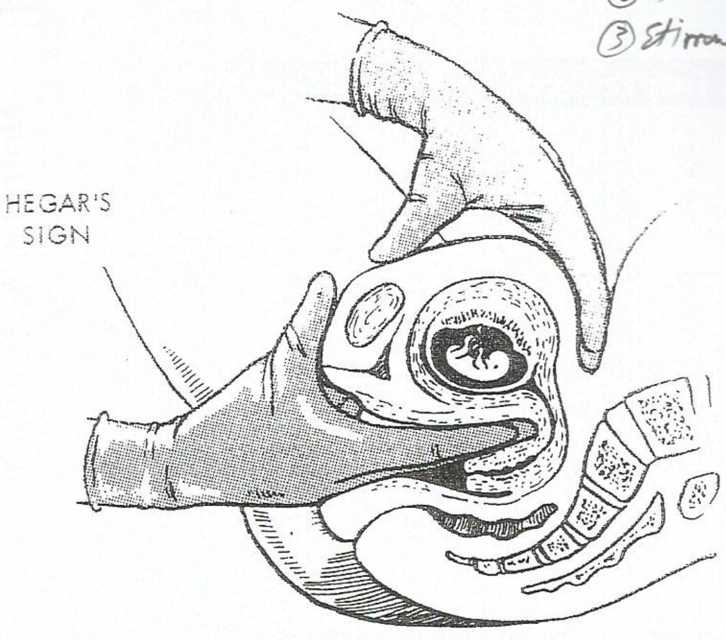
❺ Nervous system

- **Insomnia** d.t. the large uterus, leg cramps & backache
- **Carpal tunnel syndrome:** d.t. edema → disappears 2 wks after delivery
- **Placidity (calmness) & drowsiness:** d.t. ⇧ progesterone

❻ Cardiovascular symptoms

- **Varicose veins: treated by :**
  - Patients should sit with their feet elevated whenever possible
  - Nylon elastic stockings should be put in on the morning before getting out of bed..........to be removed on sleeping
- **Headaches, palpitations & fainting**
- **Physiological edema (below knee)**

Benifits of Wt. Cond.:
① enhance Pl. blood flow. (2ry pump).
② Aid aminiotic fluid secretion.
③ Stimulate fetal movement.



KEY POINTS

1. A urine pregnancy test will often be positive at the time of the missed menstrual cycle.
2. Physiologic changes during pregnancy, mediated by the placental hormones, affect every organ system.
3. Cardiovascular changes include a decrease in systemic vascular resistance and blood pressure and a 50% rise in total blood volume.
4. Elevation in serum progesterone levels is responsible for smooth muscle relaxation in the vascular system, GI tract, and genitourinary system, leading to many of the concomitant physiologic changes.

The first nine weeks

Days	Weeks	Clinical features	Scan features
0	0	Menses	
7	1		
14	2	Conception	
21	3		
28	4	Pregnancy test positive [menses due]	Empty uterus
	5		Gestational sac (hCG >2000 IU)
	6	Nausea Breast tenderness	Yolk sac, Fetal heartbeat on transvaginal scan Fetal pole 4 mm
	7		Fetal pole 10 mm
	8		Fetal heartbeat on transabdominal scan Fetal pole 14 mm
	9		Fetal pole 22 mm

Diagnosis of pregnancy

In the 1st trimester

▶ **Symptoms**

- **Amenorrhea** (NOT A SURE SIGN)
  - may have amenorrhea due to other causes
  - may have bleeding in early pregnancy
- **Breast symptoms** as heaviness, pain, enlargement, colostrum
- **Morning sickness**.....**Appetite changes**...**Frequency of micturition**
- Some ladies may experience *fatigability* & *sleepiness*, while others may have *irritability* & *insomnia*

▶ **Signs**

- **Breast signs**
- **Genital signs**
  - **Vulva** (soft & violet)............**Jaque-Mier sign**
  - **Vagina** (soft, warm & violet)......**Chadwick sign**
  - **Cervix** (enlarged, soft & violet)...**Goodell sign**
  - **Uterus** → * Enlarged & soft
    * Change in shape
    * ....**Palmer sign**
    * ....**Hegar sign** (d.t. softening of isthmus)

    Two fingers between the ant. vaginal fornix & abdomen behind the uterus can be approximated (between 6-12 wk)
    . < 6 wks → uterus is not soft enough
    . > 12 → the baby occupies the whole uterine cavity

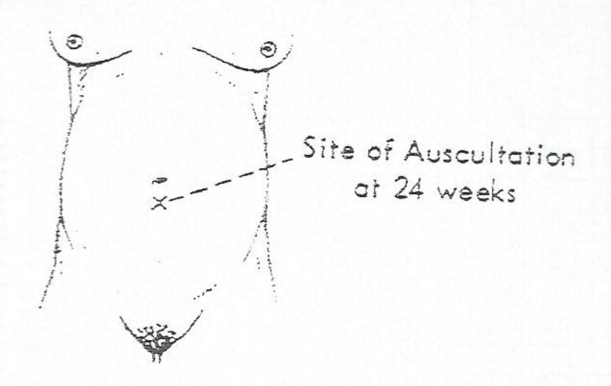
▶ **Investigations**

- **Pregnancy tests**
  * Immunological [biological are obsolete]
    - Urine tests (conventional pregnancy tests) ⇨ Latex & ELISA
    - Serum test: β-subunit (most sensitive) ⇨ RIA
- **Ultrasonography**
  - Transvaginal U/S ⇨ 5 wks (white ring)
  - Transabdominal U/S ⇨ 7 wks
  - Detection of cardiac activity ⇨ 8 wks
  - Doppler (Sonicaid) ⇨ 10 wks

♦ **Biochemical pregnancy** means detection of +ve β-HCG before missed period

♦ **The window gap**

The gap (2 weeks) between......*Biochemical* pregnancy (3rd wk) & *TVUS visualization* of pregnancy (5th wk)

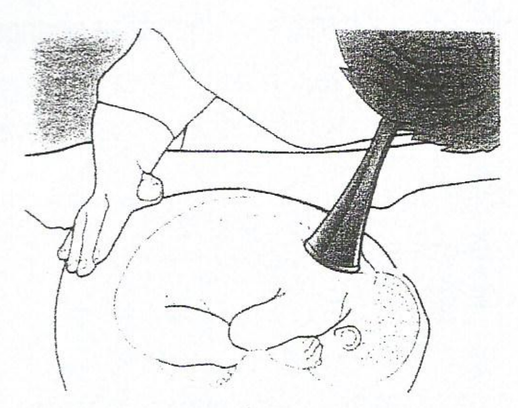




A Pinard stethoscope.

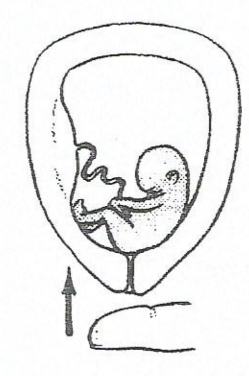
History

Pinard's stethoscope was invented in France in 1816 by René-Théophile-Hyacinthe Laennec (1781–1826). It consisted of a wooden tube and was monaural. His device was similar to the common ear trumpet, a historical form of hearing aid; indeed, his invention was almost indistinguishable in structure and function from the trumpet, which was commonly called a 'microphone'.

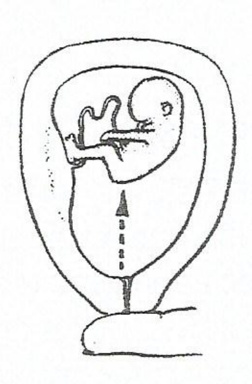


Using the Pinard stethoscope.

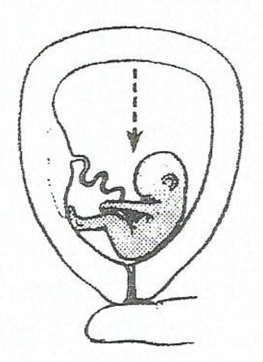




Tap gently upwards and hold finger against cervix



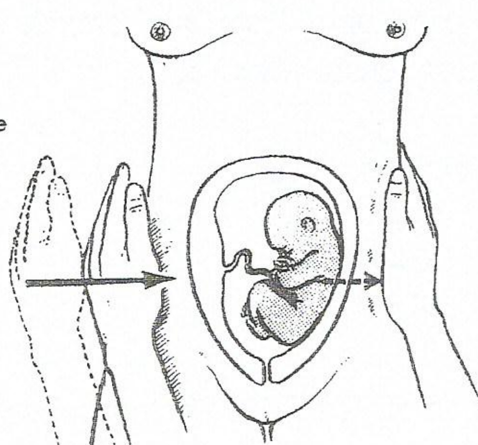
The fetus is displaced upwards



The fetus sinks and a gentle tap is felt on the finger.

24 weeks EXTERNAL BALLOTTEMENT

One hand taps the abdomen and sends the fetus across the uterine cavity.



The other hand lying on the uterus perceives the impulse.

In the 2nd trimester

▸ **Symptoms**

- Amenorrhea……….Breast symptoms increase
- **Quickening** (1st perception of fetal movement)
  - In PG → 18 – 20 weeks
  - In MG → 16 – 18 weeks
- **Progressive abdominal enlargement**

▸ **Signs**

- **Breast signs**
- **Uterine signs**
  - * **Braxton Hick's contractions**
  - * **Uterine soufflé may be heard** → soft blowing murmur
    Synchronous to the maternal pulse (due to increased blood flow through the dilated uterine arteries)
- **Fetal signs**
  - * **Ballottement** (due to movement of fetus within amniotic fluid)
    - Internal ballottement → between 16 – 28 weeks
    - External ballottement → > 24 weeks
  - * **Palpation** of fetal parts (after 24 weeks)
  - * **Inspection** or palpation of fetal movement
  - * **Auscultation** of……fetal heart sounds by Pinard stethoscope (18 wk ¤)
    ……Umbilical soufflé (funic soufflé) → Soft whistling sound
    Synchronous with the fetal heart sounds. It is due to flow of blood in the umbilical vessels and is heard sometimes when a loop of cord is in a close proximity to the anterior uterine wall

▸ **Investigations** (as in 1st trimester) → less needed as diagnosis is usually easy

Sure signs of pregnancy

- INSPECTION OF FETAL MOVEMENT
- PALPATION OF FETAL MOVEMENT / PARTS
- AUSCULTATION OF FETAL HEART SOUNDS OR UMBILICAL SOUFFLÉ
- ULTRASONOGRAPHY OR X-RAY TO VISUALIZE THE FETUS

DD of pregnancy: causes of:

- Amenorrhea
- Symmetrical enlarged uterus
- Don't forget pseudocyesis

Anternatal Care

Antenatal care

☆ Objectives

- To try to get a healthy mother & newborn
- Estimation of gestational age & expected delivery date
- Early detection & treatment of any diseases during pregnancy
- Early detection of congenital fetal malformations

☆ Consists of

- History taking
- Physical examination
- Investigations → Routine & screening tests
  Other investigations according to findings
- Plan for a schedule for return visits
- Instruction & advice
- Reassurance
- Plan for delivery

High risk pregnancy

Aim of ANC is to detect or suspect any conditions that may lead to maternal or fetal hazards i.e. to detect **high-risk pregnancy** ⮎

. **Pregnancy associated with increased risk**
. **Whether (maternal or fetal)**
. **Due to certain risk factors:**

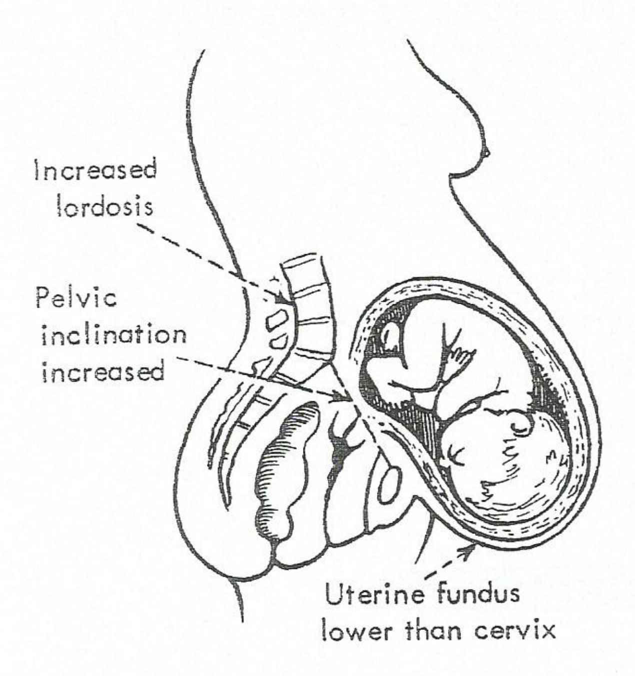
❶ *Socioeconomic*
- Socioeconomic status
- Parental occupation
- Psychological e.g. excess anxiety → preterm labor

❷ *Demographic factors*
- Maternal age (optimal age is between 20–30 yrs)
- Maternal education

❸ *Medical factors (disease)*

. PET	. Anemia	. DVT
. DM	. Renal disorders	.Thyroid disorders
. Heart disease	. Hepatic disorders	. Respiratory disorders
. Hyperemesis gravidarum		

GRAND MULTIPARITY



❶ History

▸ Personal history

- **Name**..........triple name.....
- **Age**...........lowest rate of MMR & PNMR is 20–26 yrs............

Adolescent pregnancy	Pregn. in old age (>35 yrs)
.Nutritional deficiency (immaturity)	.Nutritional def (consumption)
.Hypertensive disorders	.HTN + DM
.Dystocia (small pelvis??)	.Dystocia (osteomalacic pelvis??)
.Social & economic	.Chromosomal ... Down syndrome
++ Abortion, IUGR, PTL	

- **Marital status**
- **Parity**.................... higher MMR & PNMR in......................

1. Grandmultipara (≥ 5 deliveries) → liable to ΦΦΦ

Pregnancy	Labor
.Abortion, PTL, anemia	.Uterine atony (more fibrous tissue)
.Malpresentation (lax abd. wall)	.Obstructed labor → rupture uterus
.Placenta previa (accreta)	.PPHge
.Chronic hypertension, DM	

2. Elderly PG (≥ 35 yrs) → liable to ΦΦΦ

Pregnancy	Labor
.Abortion, PTL	.Prolonged labor (⇧ed maternal anxiety & abnormal ut. action)
.Chromosomal anomalies (Down)	.Rigid perineum → episiotomy
.Hyperemesis gravidarum	.Higher rate of CS
.PET → P.abruption, DM	

- **Address** ⇨ social conditions & environment
- **Occupation** ⇨ certain occupations have certain risk e.g.
  - Medical personnel → infectious diseases, anesthetic gases
  - Others → radiation (factories)
- **Special habits**
  - * Smoking ¤ → abortion, IUFD, IUGR, perinatal death, APHge, oligoamnios
  - * Alcohol ¤ → abortion, IUFD, IUGR, perinatal death, CFMF, mental handicap
  - * Narcotics → fetal depression & addiction
  - * Pets → risk of toxoplasmosis

▸ Menstrual history

- LMP → important for dating of pregnancy (EGA) & calculation of EDD
- Must know if it is average, regular, if sure of dates or not,
  if pregnant on period of amenorrhea, or after COC

▸ Obstetric history

Number	. Prolonged period of 2^{ry} infertility
Year of birth	. Rapid succession → liability to malnutrition
Place of birth	Previous uncomplicated home deliveries → reassuring
Antepartum period	. Repeated hypertension → expect recurrence . Previous DM → screen for DM . Previous APHge or PROM → may recur
Duration of preg.	Previous PTL → suspect maternal or uterine disease
Onset of delivery	Spontaneous......induced
Mode of delivery	Easy vaginal delivery → expect another - If previous complicated → plan for possible CS - Forceps or ventouse → suspect CPD Cesarean section → why?
Postpartum comp.	PPHge
Baby	. Alive, incubated, malformed, dead . Male / female . Weight . Breast / bottle fed
Puerperium	Puerperal sepsis, DVT

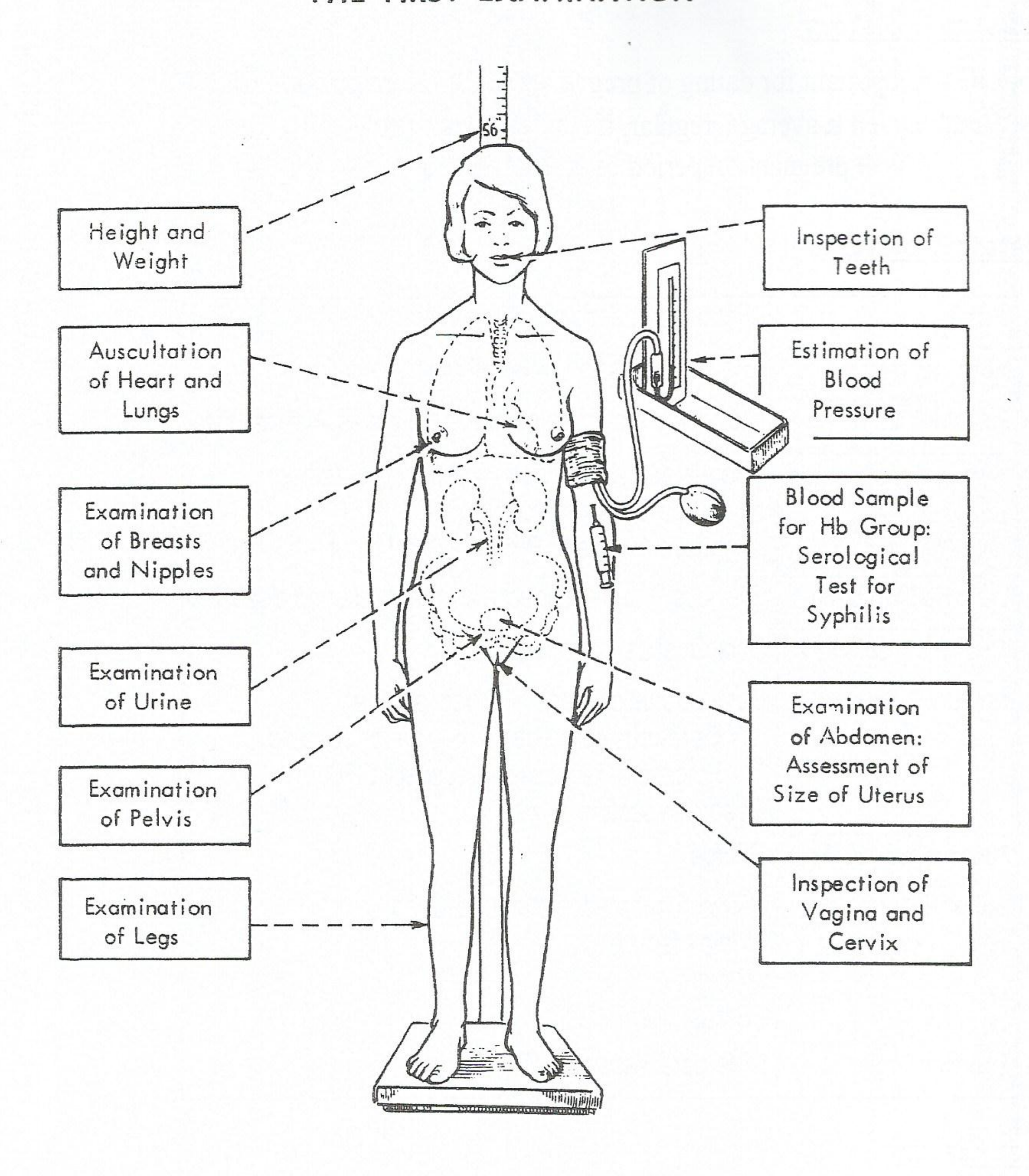
▸ Past history

- Medical → DM: screen......Hypertension: investigate..... DVT: prophylaxis
- Surgical→ Previous operations
- Drugs → May affect pregnancy or fetus e.g. oral anticoag.
- Previous blood transfusion
- Presence of allergy to drugs

▸ Family history

- DM → screen
- Hypertension → investigate
- CFMF → screen
- Twins → suspect

THE FIRST EXAMINATION



History

Nikolai Sergeivich Korotkov (1874–1920) was a Russian physician who devised a method of measuring diastolic blood pressure by applying the stethoscope to the brachial artery during the deflation of a sphygmomanometer cuff.

② Examination

▸ General قانون الادرس

- **Decubitus**: dyspnea
- **Height**: if less than 150 cm → be aware of CPD→ Cephalopelvic disproportion
- **Weight**: if obese beware of D.M., hypertension, macrosomia & dystocia

* Normal weight gain = 12.5 – 13 kg

* Underweight women = BMI < 20 kg/m^2

* Overweight women = BMI > 26 kg/m^2

Fetus	3500
Maternal fat	3500
-Blood	1500
-Extravascular fluid	1500
Uterus	1000
Amniotic fluid	1000 → maximum amniotic fluid at 38 w. then ↓ due to Pl. calcification.
- Placenta	500
- Breasts	500
Total	**12.5 – 13 kg**

☆ **3 vital data**

- **B.P.**...............for hypertensive > 140/90.....how??
- **Pulse**............abnormal pulse, esp. in heart disease
- **Temperature**...esp. in infection or PROM

☆ **3 colors**

- **Jaundice** ....pregnancy associated or induced → HTN, Hyperemesis gravidarum. -Cholestasis of preg. -Acute fatty liver
- **Cyanosis** → Eisenmenger $.
- **Pallor** .....anemia, bleeding with pregnancy

☆ **3 neck**

- **Thyroid**
- **Vessels**.....engorged normally (↑ blood volume)
- **LNs**

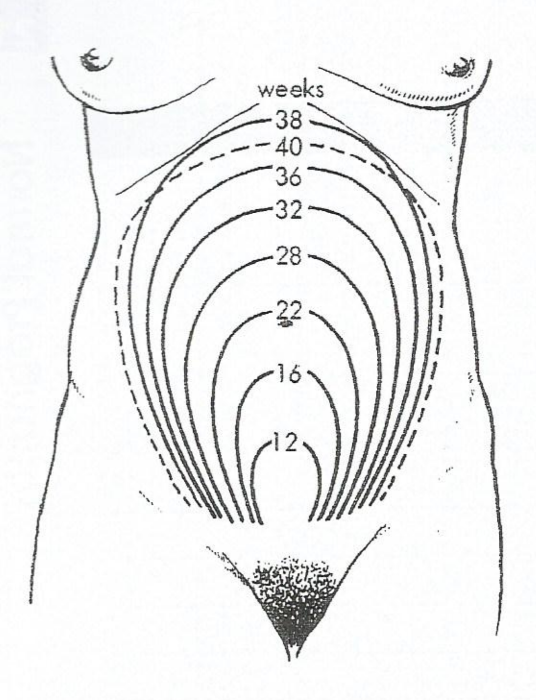
☆ **3 chest**

- **Chest** ....chest infection, PVC
- **Heart** ....what are sure signs of pregnancy?
- **Breast** ....normal changes in pregnancy, galactorrhea

(margin note: زي الباطنة بالضبط)

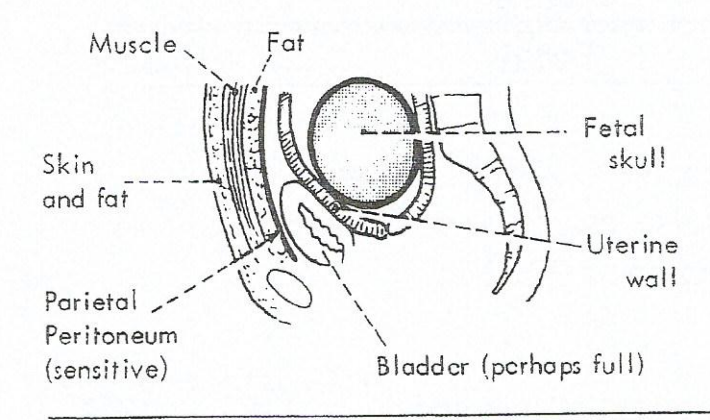
☆ **3 others**

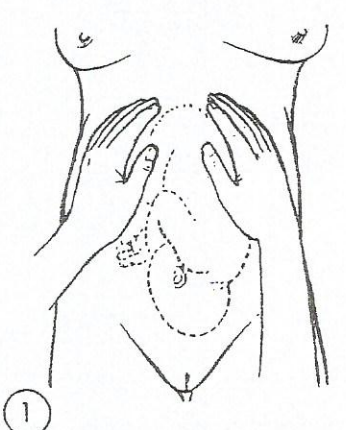
- **Gait** (look for limping → CPD)
- **Back**
- **Lower limb** for varicosities, DVT and edema.



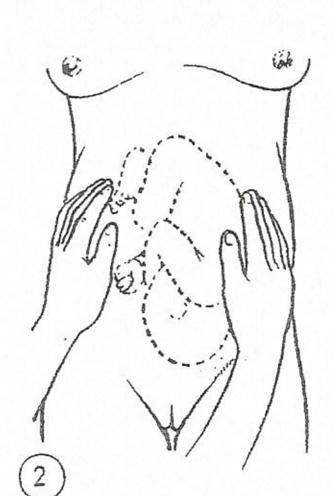
This examination must be made systematically.

Remember that the following tissue layers may interpose between your fingers and the fetal head.

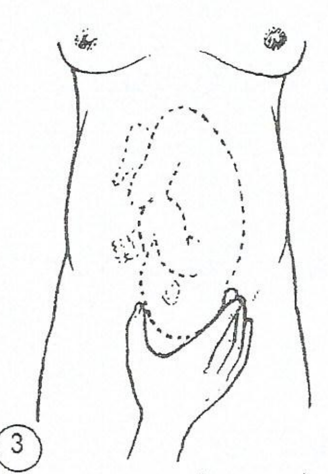




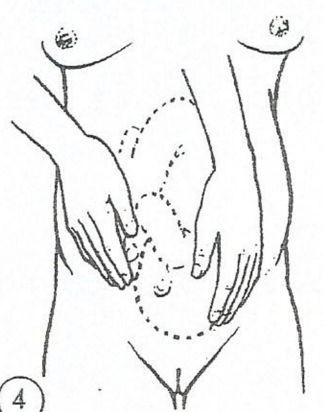
1 The fundus is palpated and the breech identified.



2 The hands palpate the contours of the uterus, identifying the back and the limbs.



3 The head should be palpated, and it should be noted whether it is mobile or fixed in the pelvic brim.



4 The examiner faces the patient's feet and gently pushes two fingers into the pelvis. This is the best method of palpating the fetal head and determining whether it is fixed or mobile.

ABDOMINAL PALPATION

- in PG : engagement occurs at 36 w, due to strong abdominal wall muscles.
- In MG : " " at time of delivery, (weak " " ")

▸ Abdominal

1. Inspection

Size → huge (twins or polyhydramnios)	Movement with respiration
Shape → if pendulous in PG → cont. pelvis	Pigmentation → linea nigra
Striae, veins, scars	Hernial orifices
Supra-pubic hair → feminine / masculine	Umbilicus

2. Palpation (4 Leopold's maneuver)

☆ Fundal level by hand or in cm above S.pubis

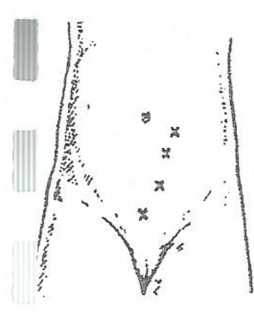
☆ Fundal grip
- Cephalic or breech
- Empty…… transverse lie

☆ Umbilical grip
- For lie
- For back & limbs
- For amount of liquor
- Expected fetal weight
- For any local uterine swelling

WEEKS	LEVEL
12	Symphysis pubis
16	
20	
24 (20-22)	Umbilicus
28	
32	
36	Xiphi-sternum

☆ 1st & 2nd Pelvic grip (Pawlick's grip)
- To determine presenting part (head, breech, empty in tr. lie)
- To determine head engagement
- To determine degree of flexion of the head e.g. extended in face

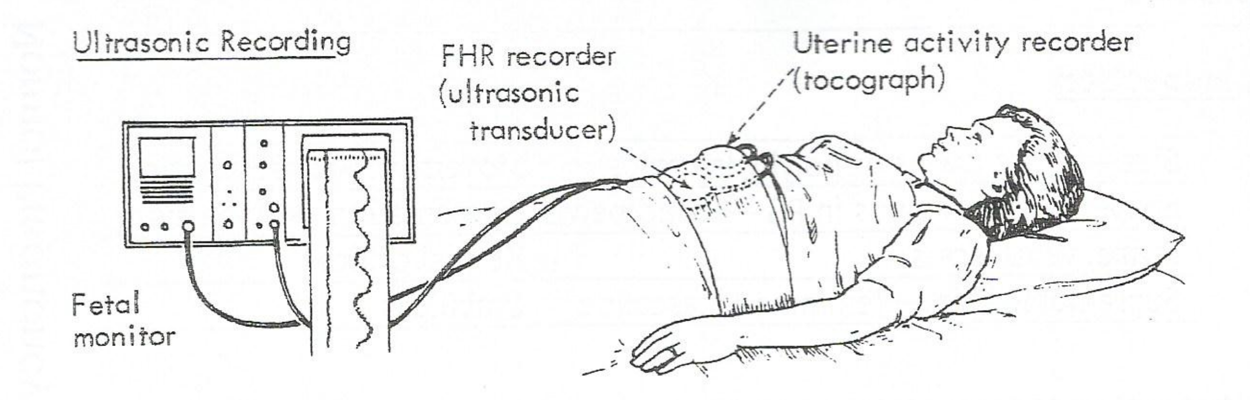
3. Auscultation



FHS can be used for	Different sites for FHS
Sure sign of pregnancy	. Normal (OA) → between umbilicus & ASIS
Fetal life / distress	. O.P. → at ASIS
Twins….Arnoux sign	. Face → MA at < umbilicus, MP at flanks
Progress in labor	. Breech → complete > umb, frank <umb
Position & presentation	. Transverse lie at one side

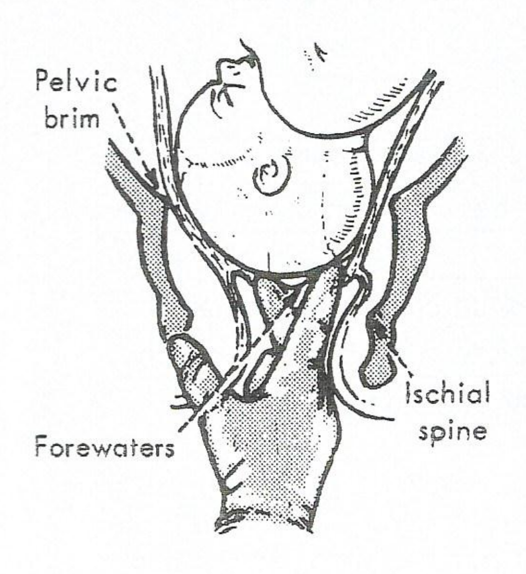
▸ Local

Done only at	In early pregnancy for	In late pregnancy for
	- Diagnosis (Hegar's sign, Ballottement)	- To diagnosis labor
	- In some complications e.g. ectopic preg	- To assess pelvis for CPD
	- Any associated pathology e.g. prolapse	- Any associated Pathology
	- To take cervico-vaginal smear	



Causes of non-engagement of head in P.G. D.

Fetal	Maternal
- Large head, Hydrocephalus	- Contracted pelvis
- Malposition or malpresentation	- Tumor in pelvis
- Multiple pregnancy	- Placenta previa
- Short cord	- Full bladder or rectum
- Polyhydramnios	- No cause may be found



③ Investigations

▸ **Routine**:

- **Blood for:** ¤
  - Blood group & hemoglobin %
  - Rh (If Rh –ve see husband.........if multipara determine if sensitized)
  - Blood sugar at 24 – 28 weeks
  - Hepatitis B surface antigen
  - Serology for syphilis
  - Antibody to rubella
- **Urine for:** Glucose.....Protein......Bacteriuria **(not C&S ¤)**

▸ **Specific**:

- **According** to history and examination
- **Ideally** → U/S ± FWB tests in high risk pregnancy

④ Return visits

- Every 4 weeks till 28 wks
- Every 2 weeks till 36 wks
- Every 1 week till delivery

• If any abnormality present → more frequent visits

• At each return visit

☆ History: Ask about any symptoms esp. fetal kicks ⤸

Dangerous symptoms ¤

....*In early pregnancy*

- Bleeding, Pain, discharge (watery or infected)
- Fever, Dysuria
- Persistent vomiting

....*In late pregnancy* (as above +)

- Symptoms of PET (blurring of vision, epigastric pain)
- symptoms of DM (polyuria, polydypsia, pruritis)
- Change in intensity or decreased fetal kicks

☆ Examination

- General ⇔ weight / blood pressure / edema
- Abd ⇔ fundal height, liquor amount, presentation, position, FHS
- PV ⇔ late or in presence of abnormality (not essential).

☆ Investigations

- Routine ⇔ . Urine in 3rd trimester for glucose, protein
  . Hb% is repeated at 34 weeks
- Specific ⇔ according to certain situations
  . α–fetoprotein (16 wk) for suspected anomalies
  . vaginal swab for chlamydia or bacterial vaginosis

Dietary advice in pregnancy

Food to avoid	Risk
Soft cheese	Listeria: fetal infection can lead to miscarriage or stillbirth
Unpasteurized milk and cheese	
Uncooked fish, e.g. sushi, smoked fish	
Unwashed salad/fruit/ vegetables	Toxoplasma: fetal infection can lead to miscarriage, stillbirth, or long-term disability
Raw and rare meat	
Unpasteurized milk	
Shellfish	Can cause food poisoning, which can precipitate premature labor
Uncooked eggs	

Dietary advice in pregnancy.

Foods that carry potential infection risks in pregnancy

Soft cheeses	Unpasteurized milk and its products may contain listeria. Those made from pasteurized milk are safe
Raw eggs	Must be avoided as there is a risk of salmonella (including puddings)
Meat or pâté	Undercooked meat may transmit toxoplasma or rarely listeria
Fruit	This should always be washed before eating as it may be contaminated with salmonella, toxoplasma or one of several intestinal parasites

⑤ Instructions & advice

- **Nutrition**
  - Caloric requirement → 2200 – 2500 K.cal / day
  - Daily increase of 300 K.cal (esp. in late pregnancy)
  - Meals should be well balanced & discourage overeating
  - If diet is adequate → no need for supplementation (except….)

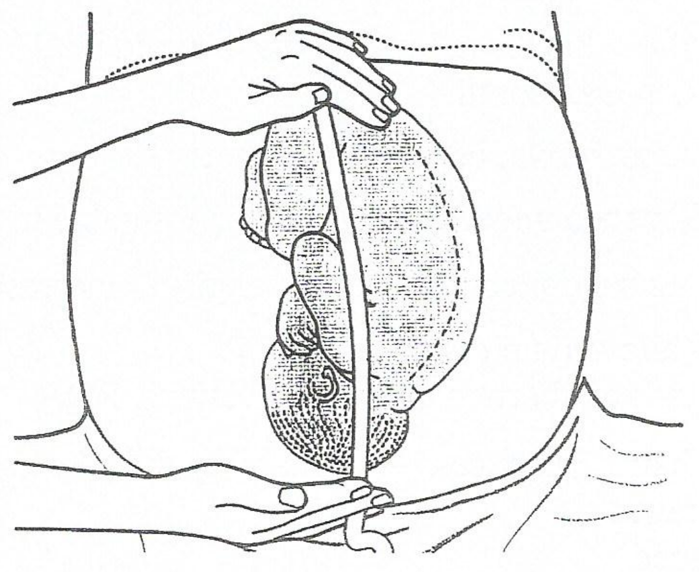
1. **Proteins**

   Requirement 1.5 g/Kg/d → addition of 1 kg protein to body weight
   Best if from animal sources (esp. milk)
2. **Carbohydrates** → to complete the caloric requirement
3. **Fats** → to complete caloric requirement
4. **Vitamins**

A → 5.000 IU /d	Ascorbic acid (vit C) → 100 mg /d
D → 400 IU /d	Vit K → PPHge & fetal Hge
B_1 → 1 mg /d	Folic acid → 0.8 – 1 mg /d ¤
B_2 → 1.5 mg /d	Nicotinic acid → 1.5 mg /d

5. **Minerals**
   - Calcium: 1 g /d (2 cups of milk)….supplementation is not essential ¤
   - Iron: 30–60 mg /d……..the only supplementation required ! ¤ ± folic acid
   - Salt: no need for either supplementation or restriction (except in HTN)

- **Rest**
  - At least 8 hrs at night & 1–2 hrs in the afternoon, better on her left side
  - Helps to increase placental flow
- **Exercise** → allowed in moderation esp walking in fresh air & swimming
- **Employment** → allowed until delivery unless physically demanding
- **Travel**
  - Allowed, but if > 6 hrs… walking / 2 hrs to avoid DVT
  - Better avoided in last month
- **Clothing** → loose unrestrictive, better no high heel
- **Bathing** → allowed & encouraged esp tub baths (less liable to accidents)
- **Douching** → high vaginal douching is condemned ¤ …increases infection
- **Sexual activity** → allowed unless there is:
  - Hemorrhage, risk of abortion or PTL, infections, ROM
- **Coffee & tea** → no harm (but excess → irritability & ⇩ Fe absorption)
- **Smoking** → discouraged
- **Alcohol** → discouraged
- **Care of teeth** → as usual
- **Medications** → should consult the obstetrician before receiving drugs.



Measurement of symphysiofundal height.

Obstetric diagnosis

- Name, Age, Para _+_, Pregnant at .... wks
- Presentation (cephalic, breech), not in labor
- Complication (obstetric...... medical)

Calculation of EDD

- **History**

1. **Menstrual delivery interval:** *'calculated from the 1st day of LMP'*
   - 280 days............**or**....40 weeks
   - 10 lunar months...**or**....9 calendar m + 7 days
2. **Naegel's rule** ¤ *'but on 3 conditions'*
   - 1st day of LMP + 7 days + 9 months **or**
   - 1st day of LMP + 7 days – 3 months
3. **Fertilization delivery interval** I know the day of fertilization.
   - Coital delivery time e.g. in IVF or rape (timed event)
   - The duration is 266 d or 38 wk or 9 m – 7 d
4. **Quickening** ⇨ PG (18–20 wk)......MG (16–18 wk)

- **Examination**

1. Fundal level Φ Φ

Causes of FL > amenorrhea	Causes of FL < amenorrhea
1- Miscalculation	1- Miscalculation
2- Pregnant on period of hge	2- Pregnant on period of amen.
3- Multiple pregnancy	3- Missed abortion
4- Macrosomia	4- IUGR
5- Polyhydramnios	5- Oligohydramnios
6- Concealed accidental hge	6- IUFD
7- Tumors: fibroids, V.mole	7- Transverse lie

2. Symphyseo-fundal height.....'McDonald rule'
   - Wks of pregnancy = height of fundus (in cm) X 8/7
3. Gravidogram
   - Progressive ↑ in FL above SP (1cm /wk after 20 wk)
4. Abdominal girth
   - Circumference around the umbilicus in inches = wks of preg
5. Pinard stethoscope detects FHS at 18–20 wks

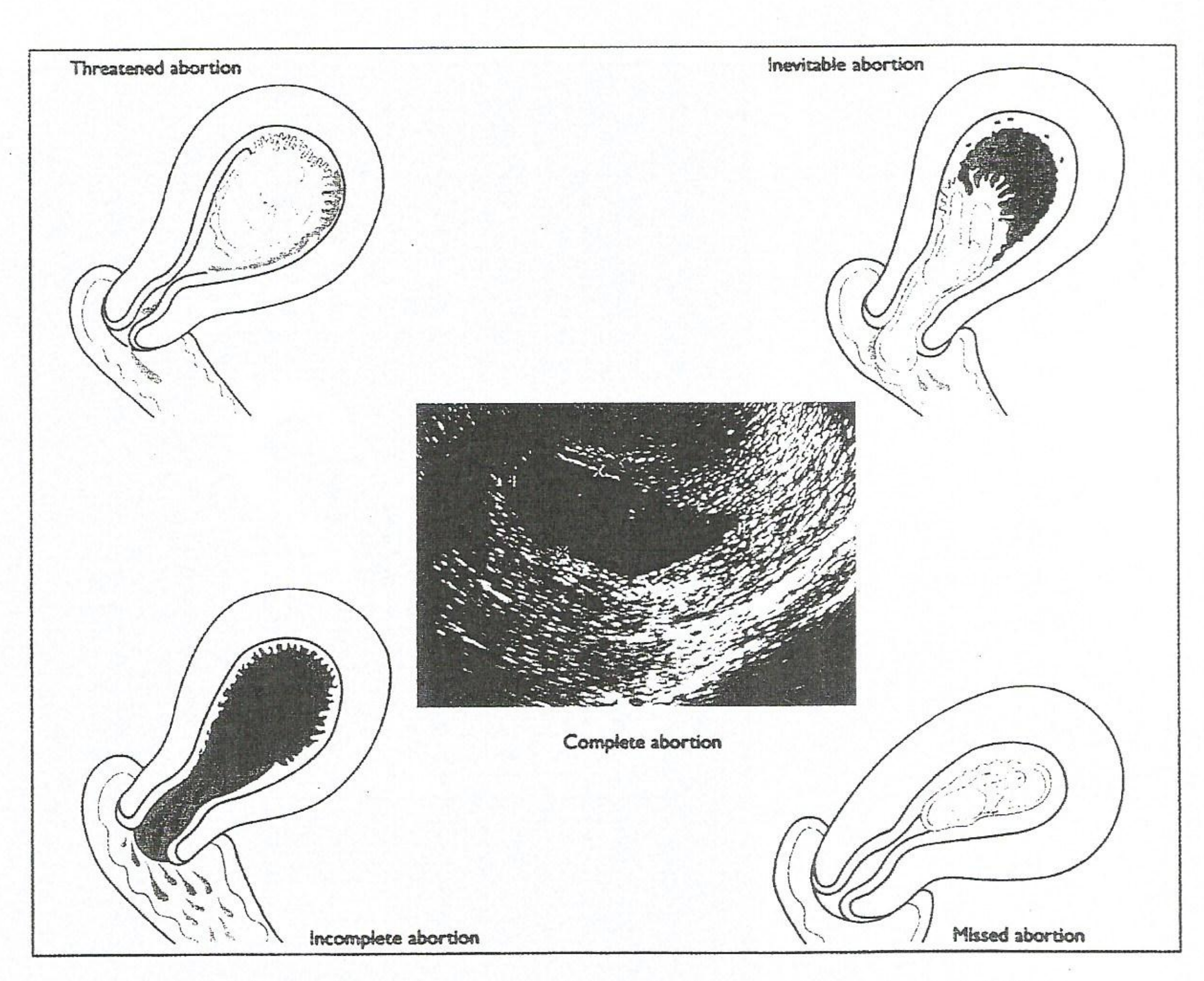
- **Investigation**

1. Ultrasound......esp the 1st trimester (the more accurate)
2. Doppler..........10 wks

Chapter 2

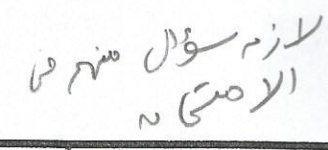
Bleeding in early Pregnancy

- Abortion
- Ectopic
- Vesicular mole



The types of abortion which may be seen. In complete abortion, the sac contains a small amount of debris.

Obstetric hemorrhage



Early pregnancy		Antepartum hge		Post P. hge
Mainly	**Others**	**Placental**	**Extra-placental**	
- Abortion - Ectopic - Vesicular mole	- Local gynecolog. conditions - Hartman's sign - Decidual hge	- Placenta previa - Abruptio placenta	. Fetal: vasa previa . Rupture uterus . Excessive show . Marginal sinus bl	- Atonic - Traumatic - Retained pl. - DIC - Acute inversion

ABORTION

Definition ➡ *termination / interruption of pregnancy* ***before*** *period of fetal viability* (gestational age at which fetus is capable of extra-uterine existence)

i.e. *(20 weeks = 500 gm)*✓✓ in developed countries & depends on the level of the NICU facilities.
or *(28 weeks = 1.000 gm)* in developing countries

Types

Spontaneous	Induced
❶ Threatened ❷ missed ⇄ ❸ septic ❹ inevitable ↳ . ❺ complete . ❻ incomplete . ❼ cervical ❽ If recurrent > 3 times → Habitual	❾ medical indication ↳ Therapeutic ❿ non-medical indication ↳ . Elective (voluntary) . Criminal

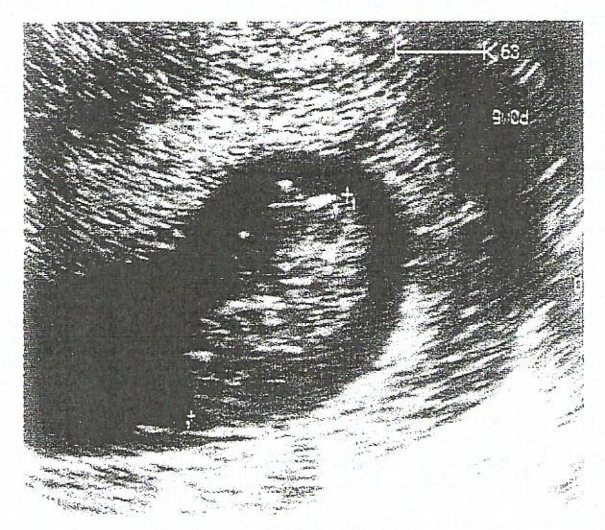
❶ Spontaneous abortion ❶

Incidence ⇨

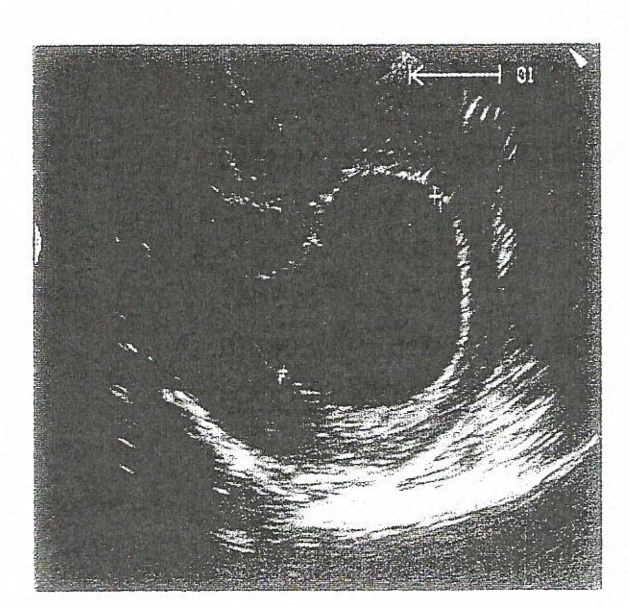
- 15–20 % ¤ ….*mostly in the 1st trimester*…..*esp. in the 3rd month* 8-12 week
  (due to some ⬇ in 'P' from C.L., while placenta still not fully developed yet; the window gap)
- True incidence *may be* much more (50–80%) due to:
  - *Subclinical* abortion (very early < patient recognition) → missed for menses.
  - *Notification* is not done in all cases (esp illegal)
- Incidence increases with ¤
  - *Increase* in maternal & paternal age
  - *Previous* abortions or stillbirth or CFMF



Intrauterine gestational scan containing a 6 mm fetal pole with a yolk sac.



An intrauterine 22 mm fetal pole, consistent with 9 weeks' gestation.



An empty gestational sac at [illegible] weeks' gestation.

Etiology

ΦΦΦ

A] Fetal (CFMF) .... malformed fetus

- The commonest cause (50–60%) ¤ of 1st trimesteric abortion
- May be ¤ → *trisomy* (50%), *polyploidy* (25%), *monosomy* x (15%)
- ***Blighted ovum*** (anembryonic sac) is a type of CFMF in which fetal tissue is replaced by *homogenous structureless* sac

B] Maternal .... macerated fetus

1. ***Maternal disease*** as hypertension or chr. renal disease
2. ***Endocrinal*** (25%)
   - ↓ progesterone → C. Luteum or placental insufficiency
   - ↑ androgens → PCO
   - Other hormonal dist. → DM, hypo- or hyper-thyroidism
3. ***Infections*** (STORCH)

   ...Any organism causing high fever e.g. typhoid fever...
   - *Bacteria* → Syphilis, mycoplasma, chlamydia, listeria
   - *Viruses* → acute viral infection, rubella, CMV
   - *Protozoa* → toxoplasma?!, malaria
4. ***Immunological***
   - *Autoimmune* → SLE, APS
   - *Alloimmune* → Rh isoimmunization
5. ***Drugs*** (cytotoxics) or ***Chemicals*** (heavy metals) or ***Radiation*** (>5 rad)
6. ***Trauma*** → direct /surgical (CL removal by mistake in appendectomy)

C] Local .... fresh fetus

1. ***Cervix*** ⇒ **Patulous internal os**
2. ***Uterus*** ⇒
   - **Congenital malformation:** septum, hypoplasia
   - **Small cavity:** submucous fibroid or Asherman's $
   - **Limited distension:** fixed RVF
   - **Overdistension:** acute polyhdramnios

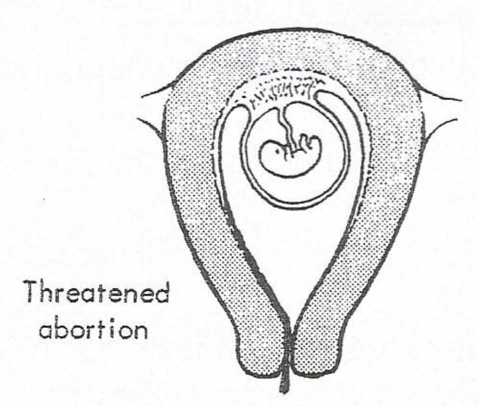
Pathogenesis

▸ **1st trimester**

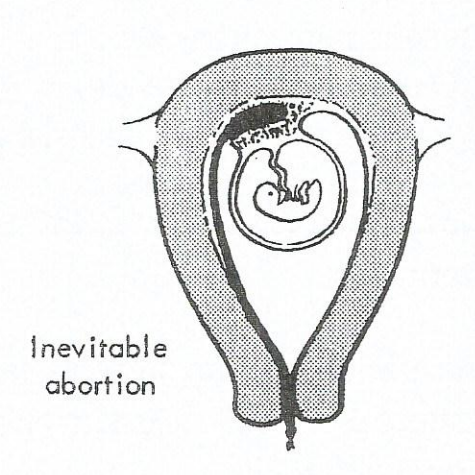
- Triad of ❶ **amenorrhea** ⇝ ❷ **bleeding** ⇝ ❸ **pain**
- Usually *bleeding* occurs into the decidua basalis (chorio-decidual hge) → uterine irritation → colic → expulsion of the pregnancy sac (either intact or as fragmented parts)

▸ **2nd trimesteric abortion**

- Usually...............amenorrhea → bleeding → pain , or
- Sometimes.........amenorrhea → **ROM** → pain (contractions)



Bleeding is slight, not retro-placental, and cervix is closed. Pregnancy is likely to continue.



CI befor 18 weeks as there is no β-receptors till 18weeks.

1) Threatened abortion

Definition ⇝ attempt of the uterus to expel the fetus
leading to *partial* separation of the fertilized ovum
with *slight* haemorrhage into the chorio-decidual space

Clinical Picture

- ○ ***Symptoms*** - Amenorrhea with symptoms of early pregnancy
  - Bleeding (slight: spotting)
  - Pain +/- (mild lower abdominal colicky pain)
- ○ ***Signs*** - Signs of early pregnancy
  - Uterus → corresponds to the period of amenorrhea
  - Cervix → closed

Investigations

- **For diagnosis (determine fetal life)**
  - **U/S** ➟ TV-US (**5** wks)…….TA-US (7 wks)…….Sonicaid (**10** wks)
  - **β-HCG** ➟ doubled every 2 days
- **For etiology** e.g. C.L. insufficiency, DM

Fate ➟
- Continues pregnancy → 70 – 80% i.e. THREATENED abortion
- Bleeding increases → INEVITABLE abortion
- Fetus dies but retained → MISSED abortion
- Infection occurs → SEPTIC abortion

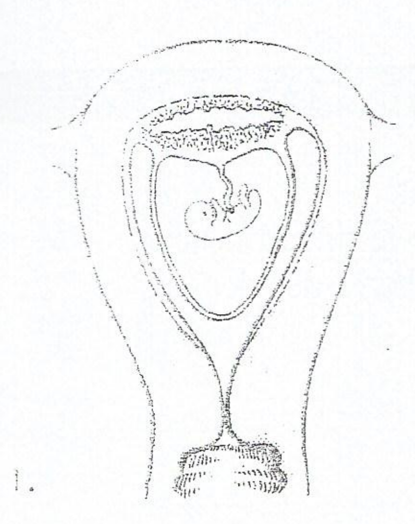
Treatment

1] Conservative

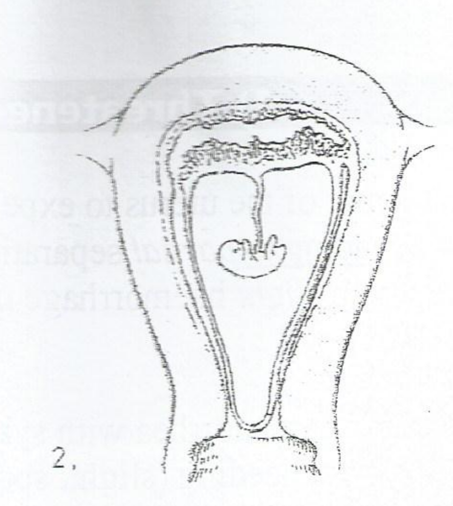
1. *Rest*
   - Physical → rest in bed till bleeding stops
   - Sexual → no sexual intercourse
   - Mental → may give sedatives as valium 5-10 mg/day
2. *Antispasmodics* e.g. antiprostaglandins
3. *Progesterone* ⇨ v.v. widely used
   - However........... .Benefit is only proven if there is well documented CLI
   - .Masking effect if missed abortion or if there are CFMF
   - .Virilization of female fetus (∴ natural forms are used)
4. *B_2 sympathomimetics* as ritodrine (more effective in 2nd trimester)
5. *Iron* & *vitamins*
6. *Anti-D in Rh-ve*

2] Terminate if

- ➢ Turned inevitable
  - Dilatation / effacement of the cervix progressively
  - Rupture of the membrane, partial protrusion of products of conception
- ➢ Turned into septic, missed

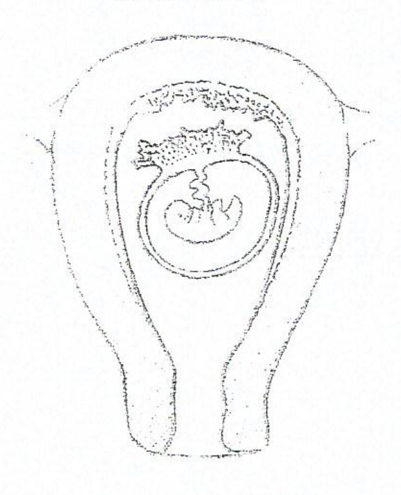


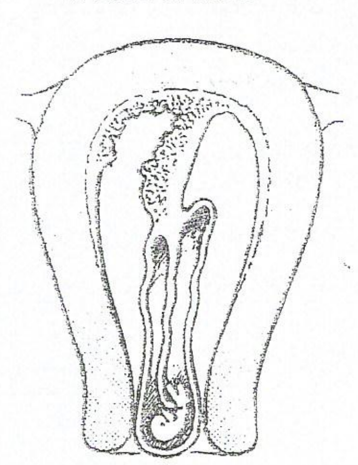
Haemorrhage occurs in the decidua basalis leading to local necrosis and inflammation.

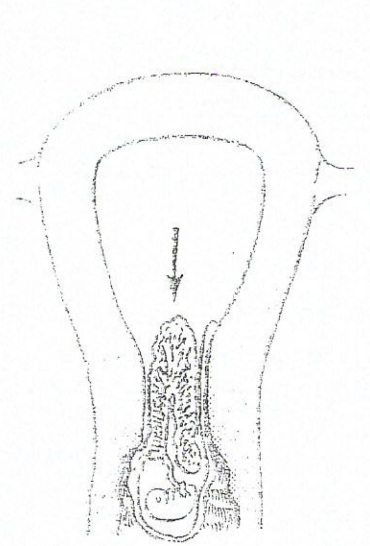


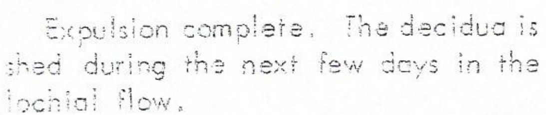
The ovum, partly or wholly detached, acts as a foreign body and initiates uterine contractions. The cervix begins to dilate.

COMPLETE or INCOMPLETE









Expulsion complete. The decidua is shed during the next few days in the lochial flow.

2) Inevitable abortion

الست بتسقط دلوقتي

- **Definition** *complete* separation of the fertilized ovum *with* progressive cervical dilatation & fetal expulsion
- **Symptoms**
  - Amenorrhea + symptoms of early pregnancy
  - Bleeding → moderate to severe
  - Pain → marked LOWER abdominal COLICKY pain (uterine contractions) with BACKACHE (cervical dilatation) = sacral pain ¤ (due to traction on utero-sacral lig).
- **Signs**
  - General → Pallor / shock (according to amount of bleeding)
  - Abdominal → uterus corresponds to period of amenorrhea
  - Local → OPENED CX (products of conception may be protruding)
- **Treatment**
  1. *Resuscitation* if bleeding is excessive
  2. *Evacuation:* 1st trimesteric → before 14 weeks → evacuation by suction or curettage
     after 14 weeks ← 2nd trimesteric → oxytocin or prostaglandins
  3. *Followed by .Ecbolics* → helps complete evacuation of remnants → (decidua).
     (Prophylactic) *Antibiotics* → reduces possibility of postabortive infection.
  4. *Anti-D if Rh –ve*

3) Complete abortion

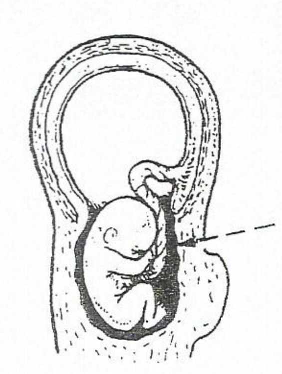
- **Definition** ⇨ all products of conception have been expelled
- **Symptoms**
  - Amenorrhea + symptoms of early pregnancy
  - Bleeding → moderate or severe
  - Pain → marked lower abdominal colicky pain with backache
    followed by expulsion of the conceptus ⇨ ⇩ ***bleeding & pain***
- **Signs**
  - General → according to amount of bleeding → may be shocked.
  - Uterus (bimanual) → smaller than period of amenorrhea
  - Cervix (P/V) → closed
- **Investigation** U/S ⇝ empty uterus
- **Treatment**
  1. Ecbolics ± Antibiotics
  2. However, some do → D&C (to ensure complete evacuation & ↓ inf)
  3. Anti-D if Rh -ve.

  - Resuscitation.

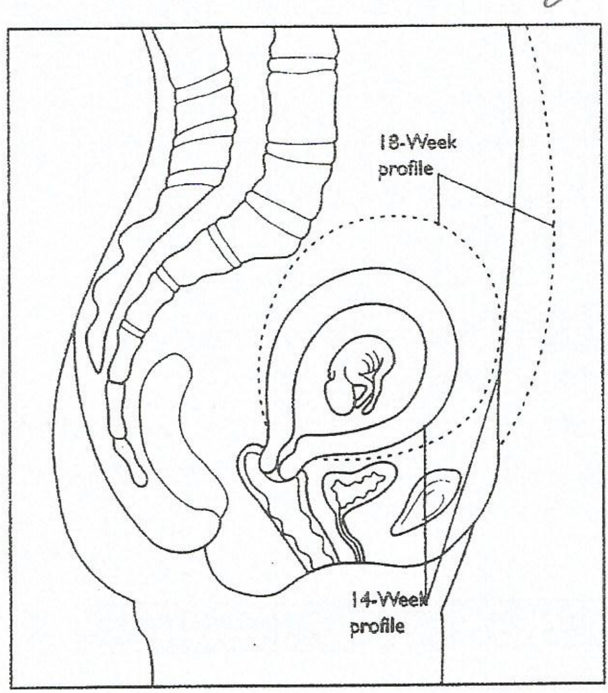
4) Incomplete abortion

- **Symptoms, Signs**
  As inevitable abortion but part of the products of conception have been expelled → therefore the uterus is < period of amenorrhea (confirm by US)
- **Treatment** → as for inevitable abortion

Cervical abortion: Contractions is able to open the internal os but not external os.



- Normally high E → stimulate pituitary → ↑ Prolactin.
- Fibrinogen level → Normally → 200-300 mg/dl
  ↳ During preg. → 400-600 mg/dl.
- DIC in missed abortion has very strange ch.ch.:
  1- Take very long time (4-6 w), only exception.
  2- The only case of DIC that can be TTT by heparin.



Missed abortion. The duration of the pregnancy is 18 weeks but the uterus has failed to enlarge beyond the size of a 14-week gestation. Note that the abdomen is flat.

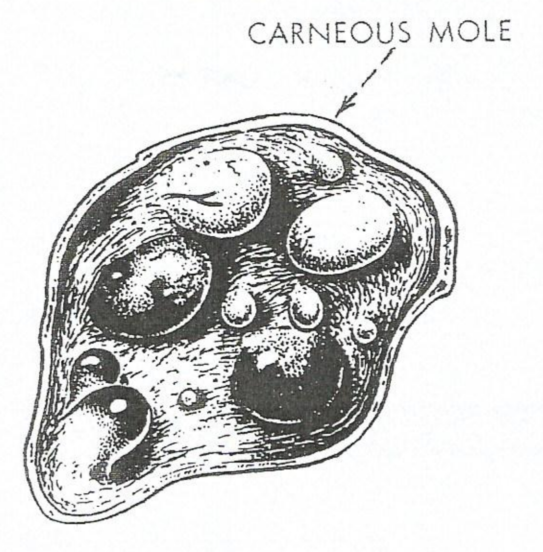
* Fresh blood: < 8 hours.
- ELISA for HCV, HIV take 24 hours.
- So fresh blood show more liability to transmitt infection.
- Inspite of that, fresh blood is much used ↔ Readily available. / Corrects hypovolemia.

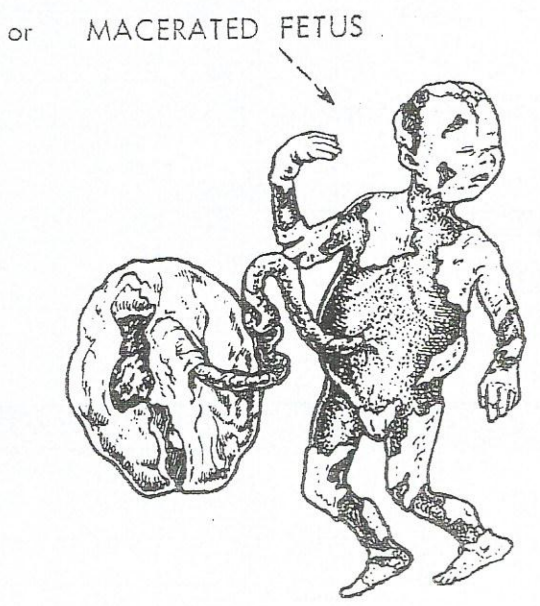
* Heparin can be used in case of single intrauterine fetal death in a twin → prophylaxis against DIC to stop the viscious circle.

When to use heparin?? 1- There is time. 2- No bleeding. 3- No operations.

If retained for long enough, the gestation may end up as a

CARNEOUS MOLE or MACERATED FETUS





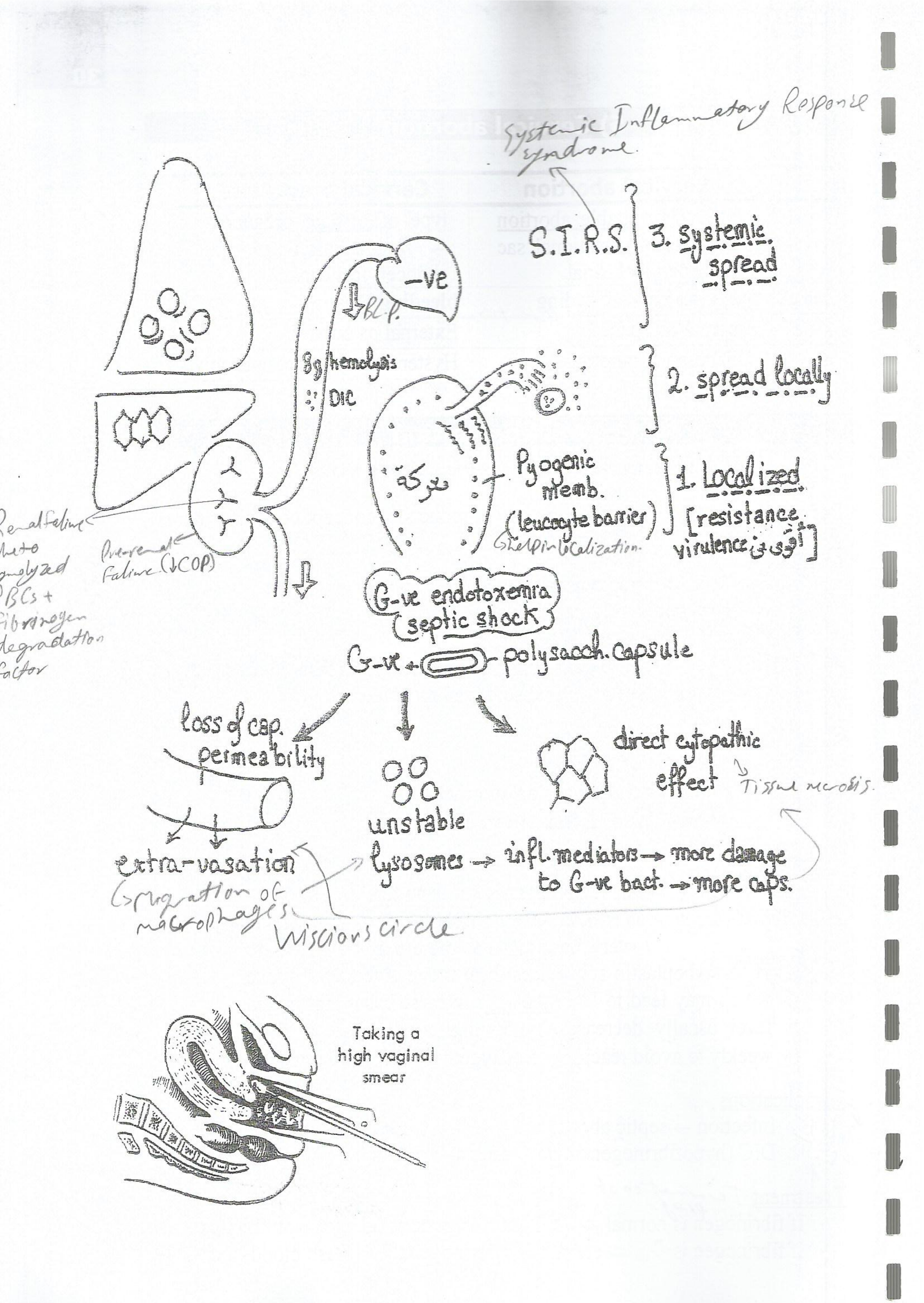
5) Cervical abortion

	Cervical abortion	**Cervical preg.** (v.rare)
Def	type of inevitable abortion → arrest of gestational sac in the cervical canal	type of ectopic pregnancy → implantation in the endocervical canal
Sympt.	**pain** (severe) > bleeding	**bleeding** > pain
Signs	Internal os opened	External os opened
ttt	Dilatation & curettage	Hysterectomy OR conservative

6) Missed abortion

(Carneous mole…fleshy mole…bloody mole)

- Definition retention of dead / non-viable products of conception within uterus
- Symptoms
  1- *Amenorrhea* ⇨ symptoms of pregnancy disappear.
  2- *Bleeding* ⇨ rarely mild dark brown (prune juice).
  . Milk secretion ¤ (d.t. ⇩ E)… it may occur normally in preg.
  . Normally E_2 blocks action of prolactin on breasts in preg.
  3- *Pain* ⇨ usually absent + absent fetal movements
- Signs
  * No general signs of pregnancy
  * Uterus → less than period of amenorrhea
  * Cervix → closed firm ± dark brown discharge ¤
- Investigations
  1- *Ultrasound* → collapsed pregnancy sac + no fetal pulsations
  2- *β-HCG* & repeat in two days for doubling
  3- *Fibrinogen level* (very important) as there may be liberation of thromboplastin substances from the retained dead tissue which may lead to DIC SLOWLY. In these cases fibrinogen level usually decreases by 50 mg/ week. ∴ it is done weekly to avoid reaching the dangerous level (100 mg/dl)
- Complications
  ○ Infection → septic abortion
  ○ DIC (hypofibrinogenemia) → after 4–6 weeks
- Treatment
  - If fibrinogen is normal ⇨ TOP (acc. to gestational age) + antibiotics
  - If fibrinogen is ⇩ed ⇨ elevate 1st (fibrinogen, FFP, fresh blood) then TOP



Systemic Inflammatory Response syndrome
S.I.R.S.
3. systemic spread
2. spread locally
1. Localized
[resistance, virulence أقوى]
-ve
BL.P.
hemolysis
DIC
معركة
Pyogenic memb.
(leucocyte barrier)
help in localization
Prerenal failure (↓COP)
G-ve endotoxemia
(septic shock)
G-ve + polysacch. capsule
loss of cap. permeability
extra-vasation
(migration of macrophages
unstable lysosomes → infl. mediators → more damage to G-ve bact. → more caps.
direct cytopathic effect
Tissue necrosis
Visciors circle
Taking a high vaginal smear

7) Septic abortion

criminal abortion is considered septic

Definition superimposed infection on any type of abortion (esp. criminal)

Organisms

- *Gram +ve* → Staph, Strept. esp Group B (GBS)
- *Gram −ve* → **E.coli**, Pseudomonas
- *Anaerobic* → **anaerobic Strept, Bacteroides,** clostridium (previously)

➢ **Source:-**

- *Exogenous*……………….instruments, sanitary pads
- *Endogenous*……………..organisms present in female genital tract
- *Hematogenous* (rare)……from a septic focus e.g. appendicitis

Clinical Picture

Symptoms

- Symptoms of abortion (amenorrhea….bleeding….pain)
- Followed by symptoms of infection
  - Fever, headache, anorexia, malaise, rigors
  - *Continuous* lower abdominal pain
  - Offensive discharge
- There may be history of a trial to induce abortion (by untrained personnel)

Signs

1- General → Toxic, pale, tachycardia, tachypnea, high fever

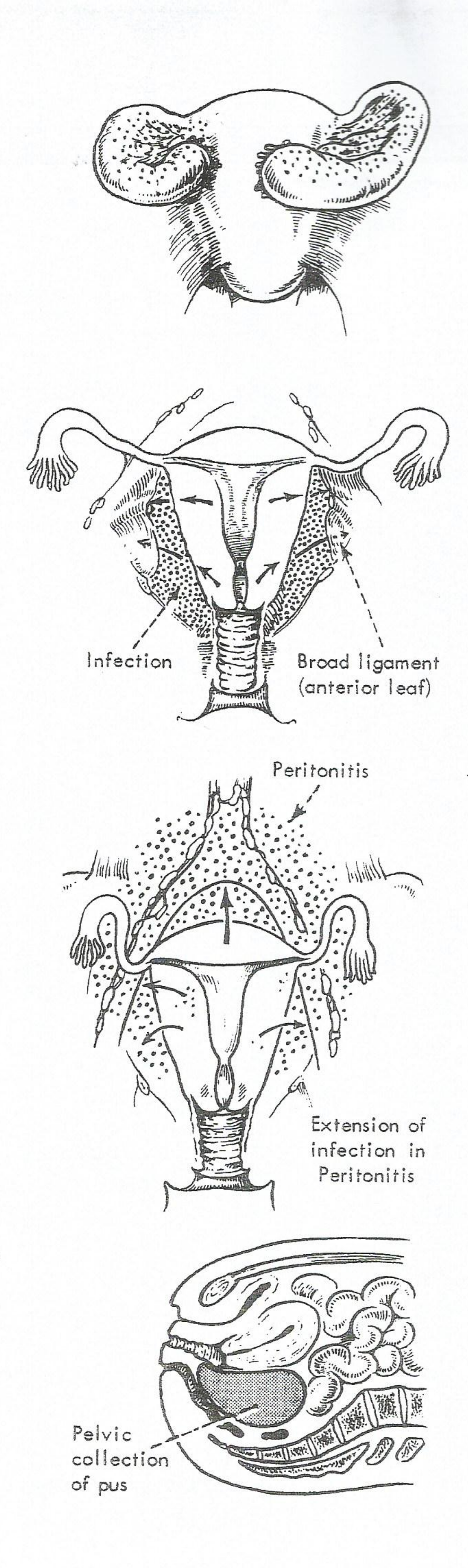
2- Abdominal
  - Decreased abdominal movement with respiration مهم؟
  - Lower abdominal tenderness & rigidity
  - Tender uterus may be felt

3- Vagina1
  - Bleeding & *offensive* discharge
  - Uterus is tender *rarely crepitations if infected* with gas *forming organisms* (physometra)
  - Fullness
  - Swelling in Douglas pouch → pelvic abscess

Investigations usually not needed, clinically diagnosed.

1. **FOR DIAGNOSIS:** U/S → dead fetus or incomplete abortion
2. **FOR ETIOLOGY**
   - Blood → CULTURE, (TLC, ESR, CRP) Acute phase reactants.
   - Endocervical or high vaginal swabs
   - X-ray → physometra (gas in uterus) + gas under diaphragm.
3. **FOR COMPLICATIONS** → renal function test & coagulation profile دول سبب الوفاة

RF DIC


Infection
Broad ligament
(anterior leaf)
Peritonitis
Extension of
infection in
Peritonitis
Pelvic
collection
of pus

Complications Φ

Local	General	Organ affection
1- Endometritis 2- Myometritis 3- Salpingitis 4- Salpingoophritis 5- Parametritis 6- Pelvic peritonitis 7- Pelvic abscess	1- Septic thrombophlebitis 2- Systemic pyaemia 3- Generalized peritonitis	1- Septic shock, ARDS 2- Acute haemolysis (esp strept & clostr) + liver affection → jaundice 3- DIC 4- Renal failure due to the above factors.

Treatment

1) Elevation of the general condition

⇨ **Antibiotics (in combination in high doses)**

- *Gram +ve* → penicillin G or cephalosporins
- *Gram–ve* → aminoglycoside as gentamycin or tobramycin
- *Anaerobic* → metronidazole or clindamycin
- *In gas gangrene* → specific antiserum

⇨ **Close observation in the ICU (in complicated cases)**

- Vital data → blood pressure, pulse, temperature
- CVP esp. in renal affection → . Urine volume
  . Repeated renal function tests
- Blood transfusion (better fresh) and intravenous fluids
- Hydrocortisone or dexamethasone →
  . ↑ tissue perfusion, stabilize lysosomes & endothelium
  . ↑ BP (+ve inotropic, restore sensitivity to catecholamines)

2) Evacuation of contents

1st trimester ➡ suction evacuation (better than curettage…why?)
- To avoid spread of infection by opening sinuses
- To avoid perforation of the soft uterus

2nd trimester ➡ * induction of abortion by oxytocin or PG
* if failed → hysterotomy
* in severe cases → hysterectomy -in toto- (esp. if old, completed her family, gas forming organisms)

3) Treat complications

1- Pelvic abscess → posterior colpotomy
2- Septic thrombophlebitis → heparin
3- Generalized peritonitis → drainage
4- DIC → fibrinogen, FFP, fresh blood
5- Circulatory collapse → vaso-pressors & sympathomimetic drugs
6- Renal failure → dialysis
7- RDS → assisted ventilation

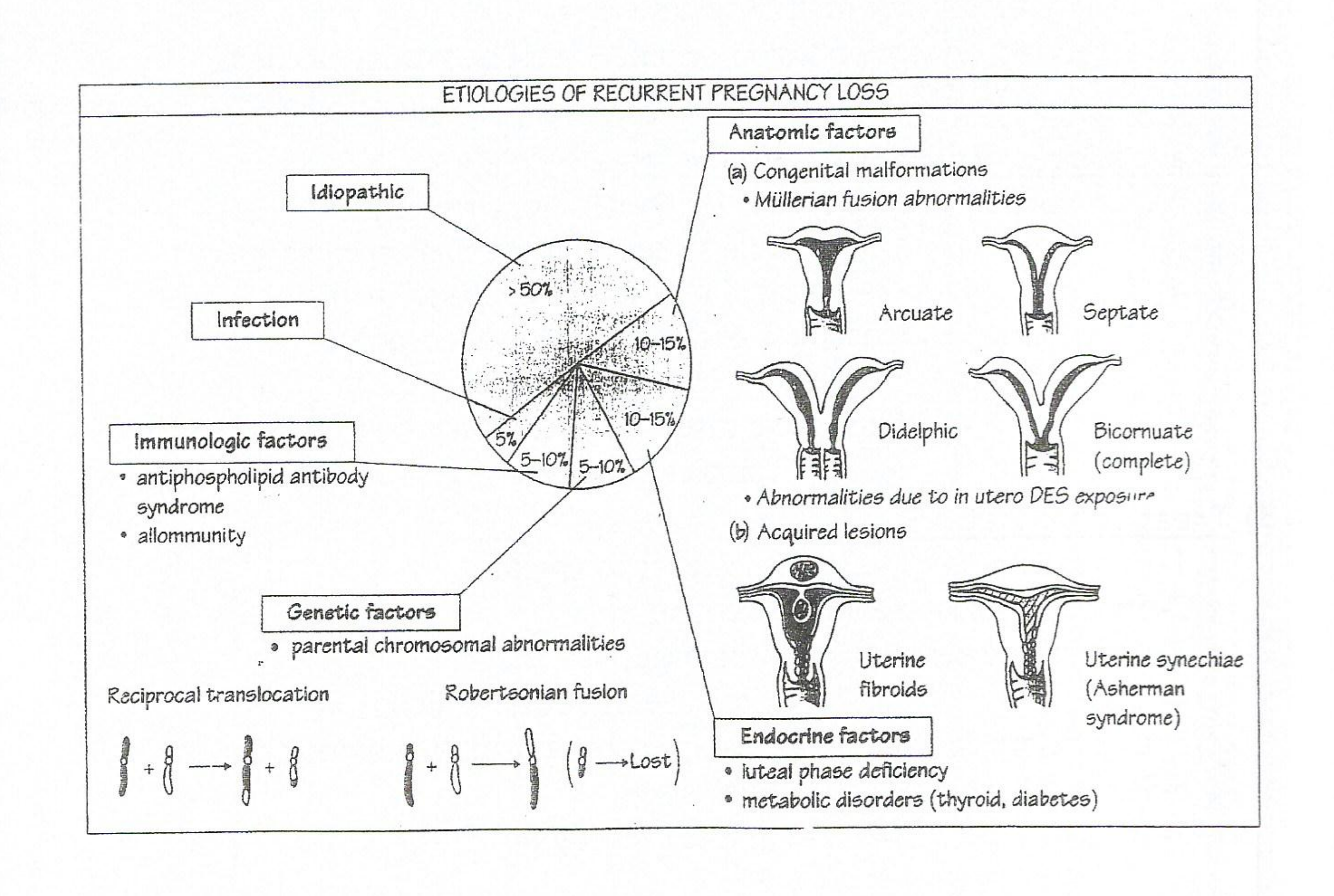
* The baby occupies the whole uterine cavity after 12 weeks

* Uterine hypoplasia: Abortion in Ascending manner.

أول مرة تسقط في الخامس واللي بعدها في السادس واللي بعدها في السابع واللي بعدها تولد في التاسع

why? 1- Mechanical stretch by the baby.
2- During Preg: ↑ E + ↑ P → stimulate the uterus to ↑ in size ⇒ E + P is used in TT of hypoplasia in non-preg. ♀

* Fixed RVF → with cerclage: baby continues to grow → anterior wall gradually becomes thinner → Ant. sacculation → uterine rupture.



8) Habitual / recurrent abortion

Definition

Three or more <u>successive</u> spontaneous abortions (some say *two*)
(If not successive it is called <u>repeated</u> abortions) (reccurrent)

Incidence

- *PG* → 10%, then percentage depends upon previous abortions:
- *Once* → 20%
- *Twice* → 26% (2–3 % of community)
- *Thrice* → 32% (< 1% of community)¤

→ ① Prog. ② Combination of Aspirin and heparin ③ Folic A. — Emperical TTT

Etiology   Φ............50% of cases are idiopathic ¤...............Φ

❶ Local causes

- Account to 30%¤ of 2nd trimesteric abortions
- Most of them is not diagnosed before multiple pregnancy losses have occurred (because they are asymptomatic)

1] **Patulous internal os** ✍
2] **CMF of uterus** → septate (25%) or bicornuate (30%) — by HSG → Give heart shape → differentiated by labaroscopy → -if fundus is present → septate uterus; -if no fundus → bicornuate.
3] **Uterine hypoplasia** → abortion in ascending manner
4] **Submucous fibroid**
5] **Fixed RVF** → abortion usually at 14-16 weeks → لأن الباطن بيكبر والرحم مش عارف يكبر
6] **Congenital Asherman syndrome** → intrauterine synechiae

❷ General causes

1- **Endocrinal** ➡ LPD......PCO.......DM.......Thyroid
2- **Immunological** ➡
  * **Autoimmune** → APS ✓✓✓ , SLE
  * **Alloimmune** → . RH incompatibility
    . Excessive HLA sharing
3- **Thrombophilia** ➡ hypercoaguable state:- ↓ protein C&S or AT_3
  factor V Leiden ↓, hyper-homocysteinemia, Ptn Z ↓
4- **Infections** ➡ e.g. toxoplasma (recently not believed to be a cause)

❸ Fetal (genetic): 4–10%¤ (structural anomalies)

* Translocation, inversion
* Mosaicism, deletion

Assessment

❶ History

➢ **Personal**

- ⇧ *Age* → chromosomal anomalies, DM, hypertension
- *Residence* → rural areas (Bilharziasis), slum areas (toxoplasmosis)
- *Occupation* → workers in heavy metal or radiation factories

➢ **Complaint** .... abortion > 3 (2) times

➢ **HPI**

- *Symptoms of abortion* → amenorrhea, bleeding, pain
- *Symptoms of complication* → fever, DIC

➢ **Past**

- *Medical* → hypertension, DM, thyroid, heart disease, ...
- *Surgery* → on cervix: trauma

➢ **Family**

- *Diseases* → hypertension, DM, ...

➢ **Menstrual**

- *Premenstrual* spotting → LPD
- *Menorrhagia* → fibroid
- *Hypomenorrhea* → hypoplastic uterus, Asherman S

➢ **Obstetric**

- *Timing* → 1st trimester is usually due to ......
  2nd trimester is usually due to ......
- *Order* → Ascending.....
  Descending......
- *Character of abortus* → - Fresh.....
  - Macerated......
  - Malformed.....
- *Special C/P* → in PIO (painless, smooth, rapid, easy)

❷ Examination

➢ **General** → medical disease

➢ **Abdominal** → swelling: fibroid uterus, ovarian swelling

➢ **Local** →

- Uterus: - Small (hypoplastic)
  - RVF
  - Bicornuate (2 bodies)
- Cervix: short, tear (PIO)

Management

Cause	Investigation	Treatment
....Patulous os ✓✓		
....Septate uterus	• If pregnant ↘ - Ultrasound - better transvaginal	.....Metroplasty (only if there are repeated failures of vaginal cerclage) as it leads to extensive adhesions ⇐
....Uterine hypoplasia	• If not pregnant ↘	.....Cyclic estrogen & progest.
....Submucous fibroid	- HSG	.....Myomectomy
....Fixed RVF	- Hysteroscope	.....Ventro-suspension (not fixation)
1- Endocrinal		
* LPD ✓	• ↓ progesterone	.....Progesterone (100 mg 1x2)
* PCO	• ↑ LH & androgens	.....Induction of ovulation
* DM	• GTT	.....Insulin
* Thyroid	• $T_{3,4}$ TSH	.....L- thyroxine
2- Immunological		
* APS ✓✓	• PTT, anti-CL, LA	.....low dose aspirin + heparin
* SLE	• $C_{3\&4}$, ANA	.....steroids
* RH incompat.	• Rh titre	.....according to titre
* HLA sharing	• HLA typing	.....immunotherapy: blocking abds
3- Thrombophilia	• Screen for protein C&S or AT_3	.....Low molecular weight heparin → anticoagulation
4- Infections	• Serum titres (rising IgM) esp. STORCH • Cervical & endomet. tissue cultures	.....Specific ttt acc to C&S
.....Genetic factors	• Family history • Karyotyping of both parents (or abortus)	.....Counseling .....Donor gametes (unethical)

**** If no cause is found (very common >50%)

- Reassurance, more periods of rest, avoid exhausting trips
- Good diet, vitamins & iron, stop smoking & alcohol
- Empirical drugs ⇨
  . *Folic acid* (3 months < & > preg. → ↓ neural tube defects)
  . *Progesterone, low dose aspirin, heparin*

Notes

CX dilatation: ↑ in width.
CX effacement: ↓ in length

Patulous internal os

- **Etiology** Φ
  - *Congenital*
    - Increased muscle tissue in cervix > 10%
    - Associated with other uterine malformations as septate, bicornuate uterus, hypoplastic uterus
    - DES (diethyl-stilbesterol) exposure in utero
  - *Acquired*
    - *** due to obstetric trauma**
      - Forceps or ventouse before full cervical dilatation
      - Breech extraction before full cervical dilatation
      - Manual dilatation of the cervix
    - *** due to gynecologic trauma**
      - Dilatation of the cervix excessively or too rapidly
      - High amputation of the cervix
      - Cone biopsy of the cervix

- **Clinical picture**
  - This condition usually leads to classic picture of
    - *Painless* effacement & dilatation of the cervix
    - Uterine contractions are late & not very painful
    - PROM followed by rapid delivery of a fresh abortus with minimal discomfort
  - The abortion or premature labor usually occurs in *descending* fashion i.e. at 7 months → 6 months → 4 months, etc.

- **Investigations**

  1] If pregnant
  - Serial U/S examination (better done transvaginally)
  - to determine length (2.5–3 cm) & width (1 cm) of internal os

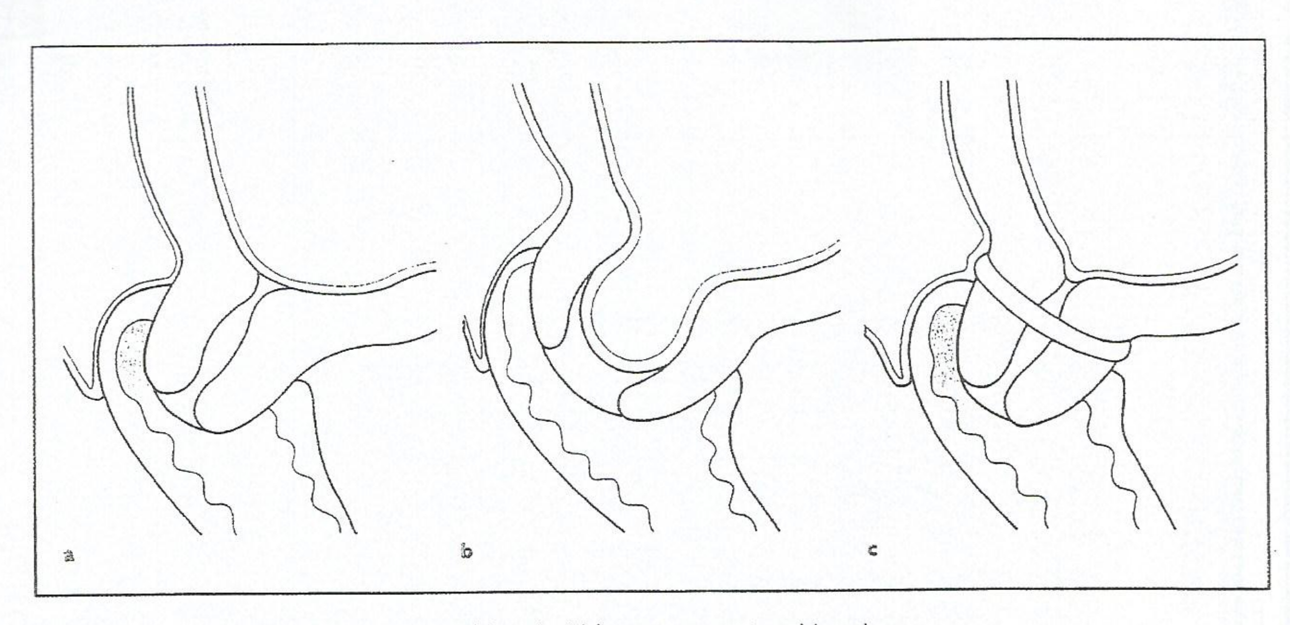
  2] If not pregnant
  - HSG → funneling (loss of uterine waist)
  - Ability to pass Hegar dilator No **8** or hysteroscope No **8** with no resistance & little pain ✗
  - Pediatric Foley catheter with 1 ml inflated balloon can be pulled through the os without resistance ✗

- **Treatment**

  1] If pregnant ⇨ *cerclage*

  2] If not pregnant ⇨ *trachelorrhaphy* (esp after cervical tears)

CS after cerclage: 1- Abdominal cerclage.
2- Shirodkar's op.



Cervical incompetence. (a) Normal cervix at 16 weeks; (b) Incompetent cervix at 16 weeks; (c) Cerclage with an unabsorbable suture.

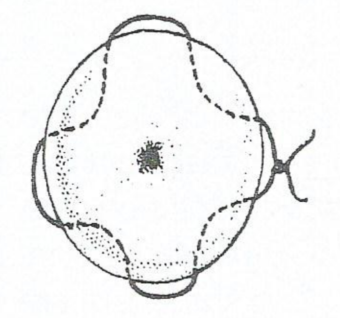
ليه مش بعد 16 اسبوع ؟ ←
1- Pathophysiology has already started.
2- Injury of amniotic ~~ft~~ membr. → PROM

ليه مش قبل 12 اسبوع ؟ ←
1- Pathophysiology has not started yet.
2- Percentage of spontanous abortion is high in 1st trimester.

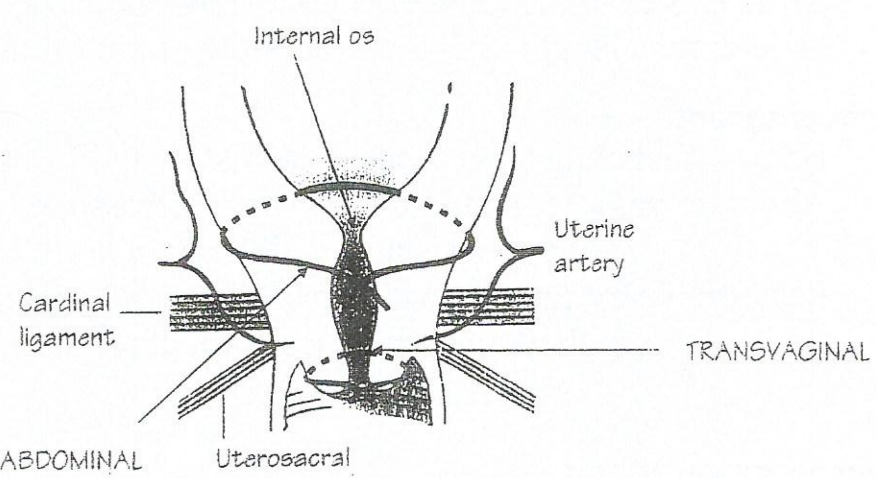
History

هندي. ← Vithal Shirodkar (1899–1971) was an obstetrician and gynaecologist from Goa who proposed a purse-string suture of fascia lata around an incompetent cervical os to prevent second-trimester loss.

Ian McDonald (1922–1990), from Australia, simplified the Shirodkar operation with the use of a silk purse-string suture around the cervix.



- Avoid 3 & 9 o'clock → to avoid injury of descending cervicovaginal a.
- Ends of tape are left long → for easy removal, hanging from post. lip of cx → ↳ post. fornix is wider.



Cerclage

▸ **Types**

1. **McDonald's** → 70% success
   . 4 bites with purse string suture (Nylon or Mersilene)
   . Taken around the highest portion of the portio-vaginalis….why?

2. **Modified Shirodkar's** → 70% success
   . The bladder is dissected upwards
   . Thus sutures could be taken at level of internal os

3. **Abdominal**
   . Indications → repeated failed vaginal cerclage or short / absent cervix
   . Delivered by → CS (permanent cerclage)
   . If failed < 28 wks → hysterotomy must be done (a great disadvantage)

> **Indication** ⇨ PIO, uterine malformation (septate), triplets
> **Insertion** ⇨ 12–14 weeks
> **Postoperative** ⇨ anti-PG, progesterone, antibiotics,
> **Removal** ⇨ 2 weeks < EDD (≈37 wks)

▸ **Complications**

- Injury to bladder
- Injury to membrane → ROM → tape must be removed
- Infection → tape must be removed & terminate
- Abortion or PTL

Antiphospholipid syndrome

▸ **Definition** . autoimmune dis. forming antibodies against phospholipid proteins
. it may be 1^{ry} (alone) or 2^{ry} (associated with CT disorders: SLE)

▸ **Diagnosed by** ΦΦ

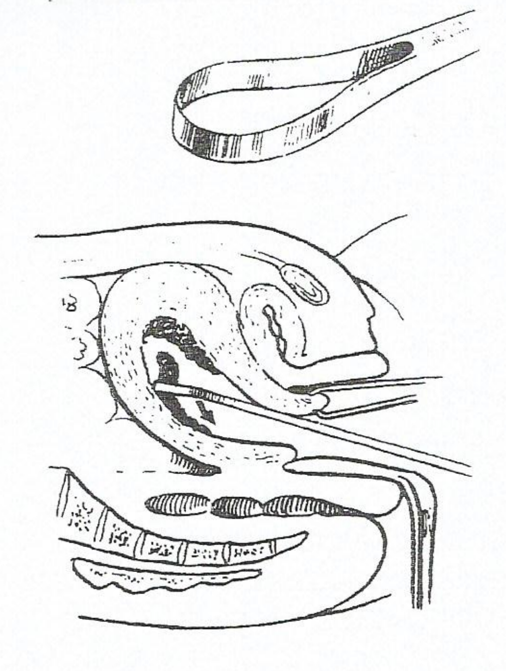
❶ Recurrent ¤¤
- Thrombosis → *arterial & venous*
- Fetal loss → . ≥ 3 consecutive miscarriages (< 10 wks)
  . ≥ 1 fetal death (> 10 wks)
  . ≥ PTL (< 34 wks) due to severe PET
- PIH → usually severe ± pl. insufficiency ± IUGR ± abruptio pl.

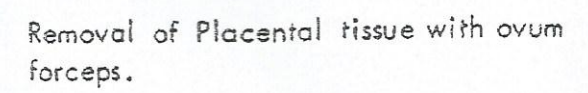
❷ Positive antibodies
- Anticardiolipin antibodies (ACA) } high
- Lupus anticoagulant (LAC) } false +ve

▸ **Treatment**

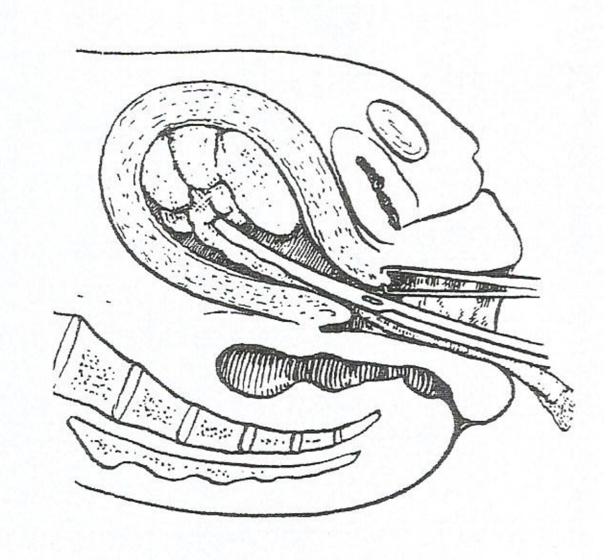
- Baby aspirin (75 mg /day) + Heparin 5.000 units SC /12 hrs
  or LMW heparin ✓ 30–40 mg /day
- Corticosteroids → not more used

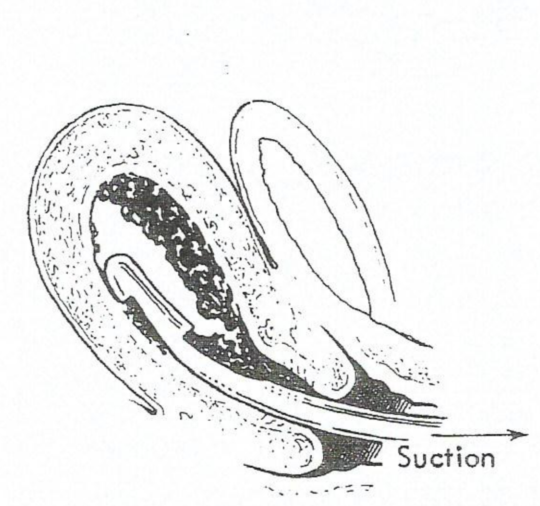
CURETTAGE. A blunt curette may be tried first but usually a sharp curette is required.

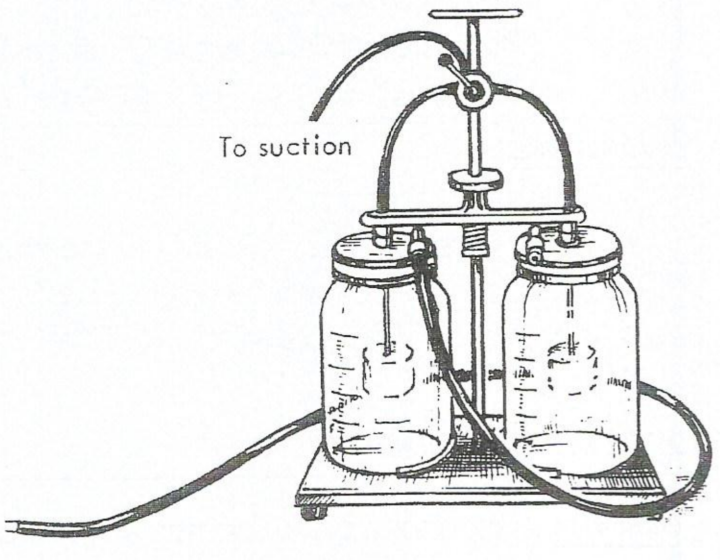


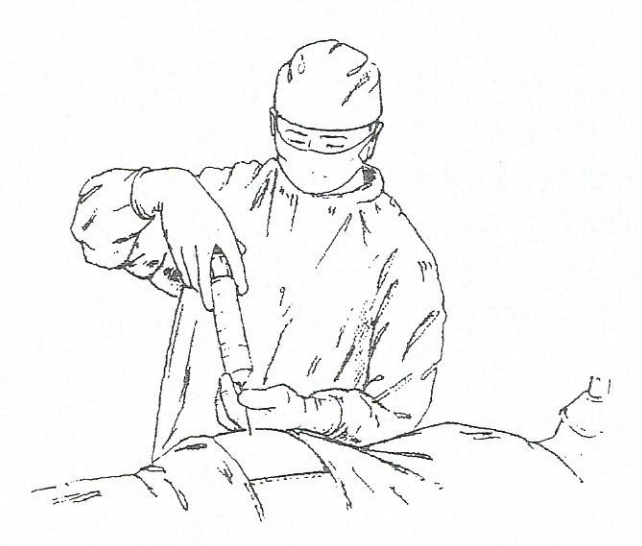


Removal of Placental tissue with ovum forceps.

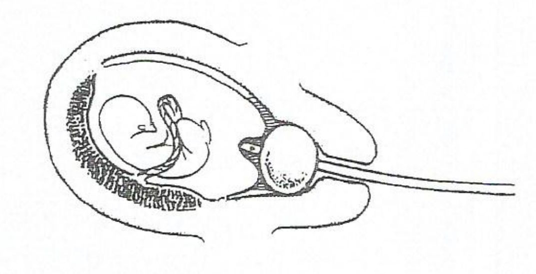








Ingection of hypprtonic sonution



Local PG
(Intrauterine-Extraamniotic)

❷------------Induced abortion-----------❷

1) Therapeutic abortion

▸ **Indications**

❶ *Maternal*

- *Medical disorders e.g.*
  - Advanced:- Heart disease / chronic HTN / renal disease
  - Active pulmonary T.B./ severe hyperemesis
- *Malignancy*
  - Genital tract / breast malignancy
  - Chemotherapy or radiotherapy
- *Mental psychological illness*

❷ *Fetal*

- Missed abortion / blighted ovum / vesicular mole
- Exposure to teratogenic agents → rubella radiation

▸ **Methods**

A- Before 14 weeks ➡ suction evacuation Or dilatation & curettage

B- After 14 weeks

- *Prostaglandins*
  - Local (intra-amniotic or extra-amniotic)
  - Vaginal or intracervical tablets
- *Oxytocin*
- *Intra-amniotic injection of hypertonic solutions* ✗✗
  - Saline 20%......Urea 30-40%.........Glucose 50%
  - Complications → danger of infection & DIC
- *Hysterotomy* if all fail or there is severe bleeding

2) Criminal abortion

▸ **Definition**: TOP for non-medical reasons (in countries where abortion is illegal)
It is called elective (voluntary) abortion (in countries where abortion is legalized)

▸ **Methods used**

1- uterine stimulation → methergine, purgatives
2- Intra-uterine manipulation to induce cervical dilatation or ROM
3- Evacuation by untrained Doctor under Septic conditions

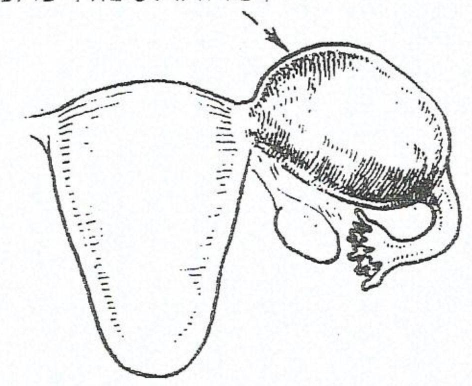
▸ **Common complications**

- Genital tract trauma e.g. uterine *perforation*
- Infection → *sepsis*

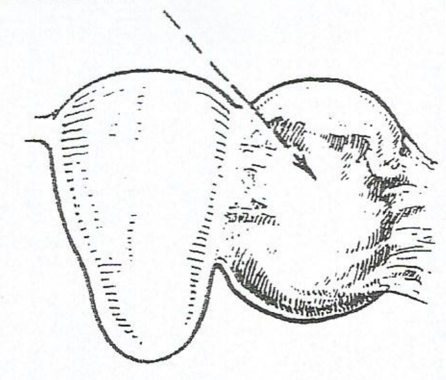
▸ **C/P & treatment** as SEPTIC abortion

ABORTION—DIFFERENTIAL DIAGNOSIS

TUBAL PREGNANCY

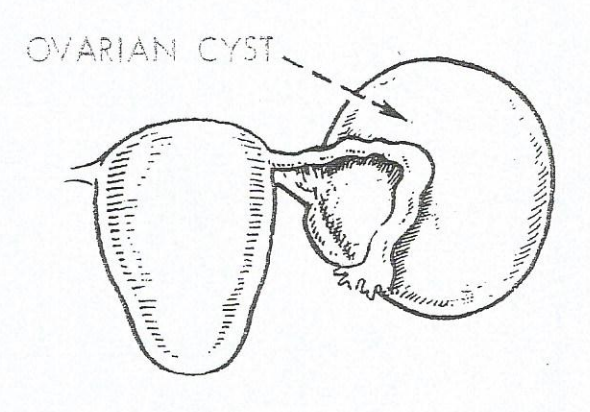


PYOSALPINX



FIBROIDS

OVARIAN CYST



METROPATHIA HAEMORRHAGICA may simulate abortion so closely that the distinction can be made only on the histological appearances.

Much uterine bleeding has no organic explanation and the patient accepts or supplies a diagnosis of abortion for want of anything better,

Abortion
- scientefic term.
- induced ← يعني بانو ايجابي.
- 1st trimester
حاجة صغيرة وقعت وما اخدتش بالي

MisCarriage
- lay people term.
- spontanous. ← يعني بانو ايجابي.
- 2nd trimester
حاجة كبيرة وقعت مني

KEY POINTS

1. The most common cause of first trimester abortions is fetal chromosomal abnormalities.
2. It is important to rule out ectopic pregnancy with history, physical examination, laboratory studies, and ultrasound.
3. First trimester incomplete, inevitable, and missed abortions are usually completed with a D&C or medical management with prostaglandins, although expectant management is also used.
4. RhoGAM should be given to all Rh-negative patients with bleeding.

KEY POINTS

1. Most second-trimester abortions are secondary to uterine or cervical abnormalities, trauma, systemic disease, or infection.
2. D&E, prostaglandins, or oxytocic agents can be used for the management of spontaneous abortions in the second trimester that need assistance to completion.
3. The risk of uterine perforation from D&E is greater in the second trimester than in the first.

Extras

➢ *What* is *medical* abortion.......?

➢ *What* is the *DD* of abortion..........? Look the figure

➢ *What* are the *complications* of abortions.....?

IMMEDIATE.........COMP. OF INTERFERENCE...........LONG SEQUELAE

➢ *What* are the causes of *postabortive bleeding*.....?

THE COMMONEST.....THE MOST SERIOUS... ... PERFORATION.....ASSOCIATION

➢ *What* is the *management* of postabortive bleeding......?

➢ *What* is the weight of the *smallest* fetus ever survived.....?

➢ *What* is the difference between term abortion / *miscarriage*....?

➢ *What* is the management of a case of *idiopathic habitual abortion*.....?

➢ *How to prevent spontaneous abortions...?*

Most abortions could not be prevented (except if there is obvious cause e.g. DM, PIO). This seems reasonable as most spont abortions are due to C.F.M.F.

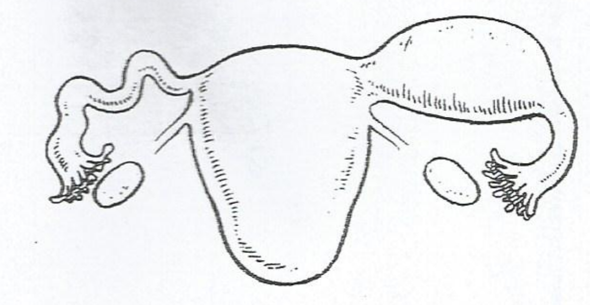
➢ *What* are the other *rare* causes of bleeding in early pregnancy...?

- LOCAL GYNECOLOGICAL CAUSES
  - Known by speculum examination e.g. ulcer, polyp, HPV, tumor
- HARTMAN'S SIGN (scanty *spotting* at time of implantation)
  - Due to erosions of some vessels 1 week after fertilization
  - Importance → wrong calculations of EDD
- DECIDUAL haemorrhage (monthly scanty *bleeding* at time of menstruation)
  - Due to separation between decidua capsularis & decidua parietalis
  - bleeding occurs till 12 weeks (until the 2 layers fuse together)

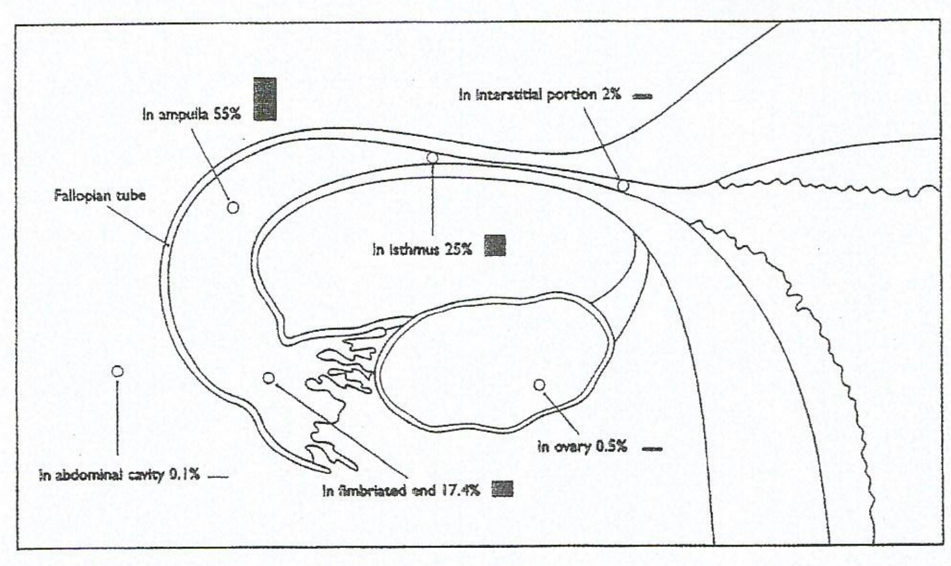
➢ What are the main etiological causes of the 2^{nd} *trimesteric* abortion....?

* Cervical incompetence & other uterine malformations
* Early fetal demise (early IUFD) e.g. syphilis, Rh (both rare now)
* Placental causes e.g. APS, circumvallate placentation
* Uterine overdistension e.g. twins, acute polyhydramnios
* Trauma & infection

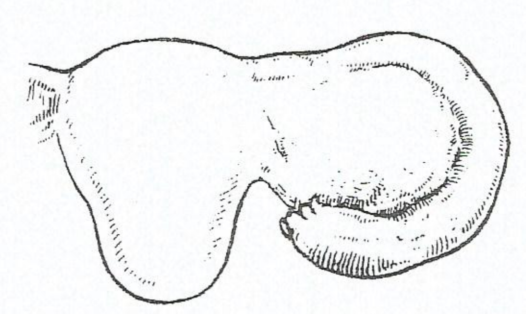
Effect of DES on ♀ genital system:
1- Vaginal adenosis → adenocarcinoma.
2- Patulous int. OS. → habitual abortion.
3- T-shaped uterus
4- Congenital tubal anomalies.
↳ hypoplasia, accessory ostia, diverticula.



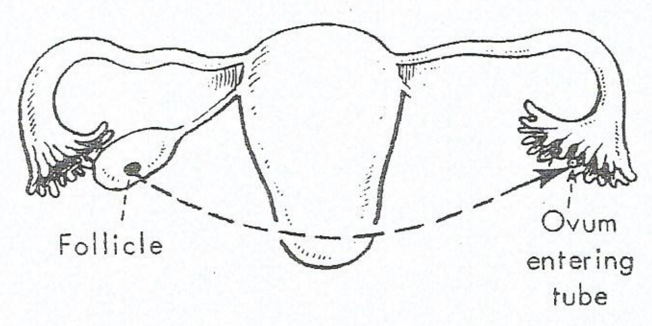
Tubal pregnancy

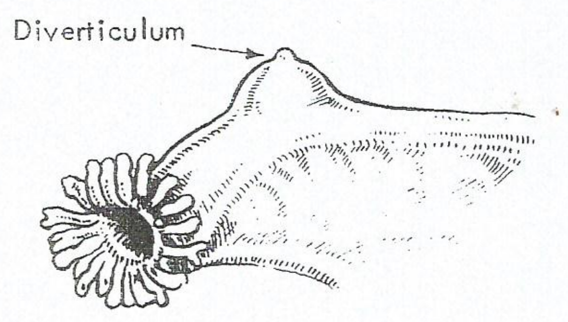


Sites of ectopic gestation implantation, with the relative frequency of occurrence.



Chronic salpingitis





Ectopic Pregnancy

Definition

- Implantation anywhere outside the ENDOMETRIAL CAVITY
- It is responsible for 10% OF MMR ¤
- INCIDENCE is ↑ed 4 folds in the last 20 yrs from 1→3 % d.t. ↑ of: ¤
  ❶ STD's ❷ contraception (IUCD) ❸ ART (IVF)

Sites

Uterine (v.rare)	*Extra-uterine*			
1- Cervical	1- Tubal 99% ✓✓			
2- Rudimentary horn	**Interstitial,**	**isthmus,**	**ampulla,** (the most wide)	**fimbria**
3- Angular = Cornual preg.	**2**	**15**	**✓ 80 ¤**	**5**
4- Intraligamentary → Broad lig.	2- Ovarian 0.5%			
	3- Peritoneal (abdominal: 1^{ry} or 2^{ry})			

Etiology ΦΦ

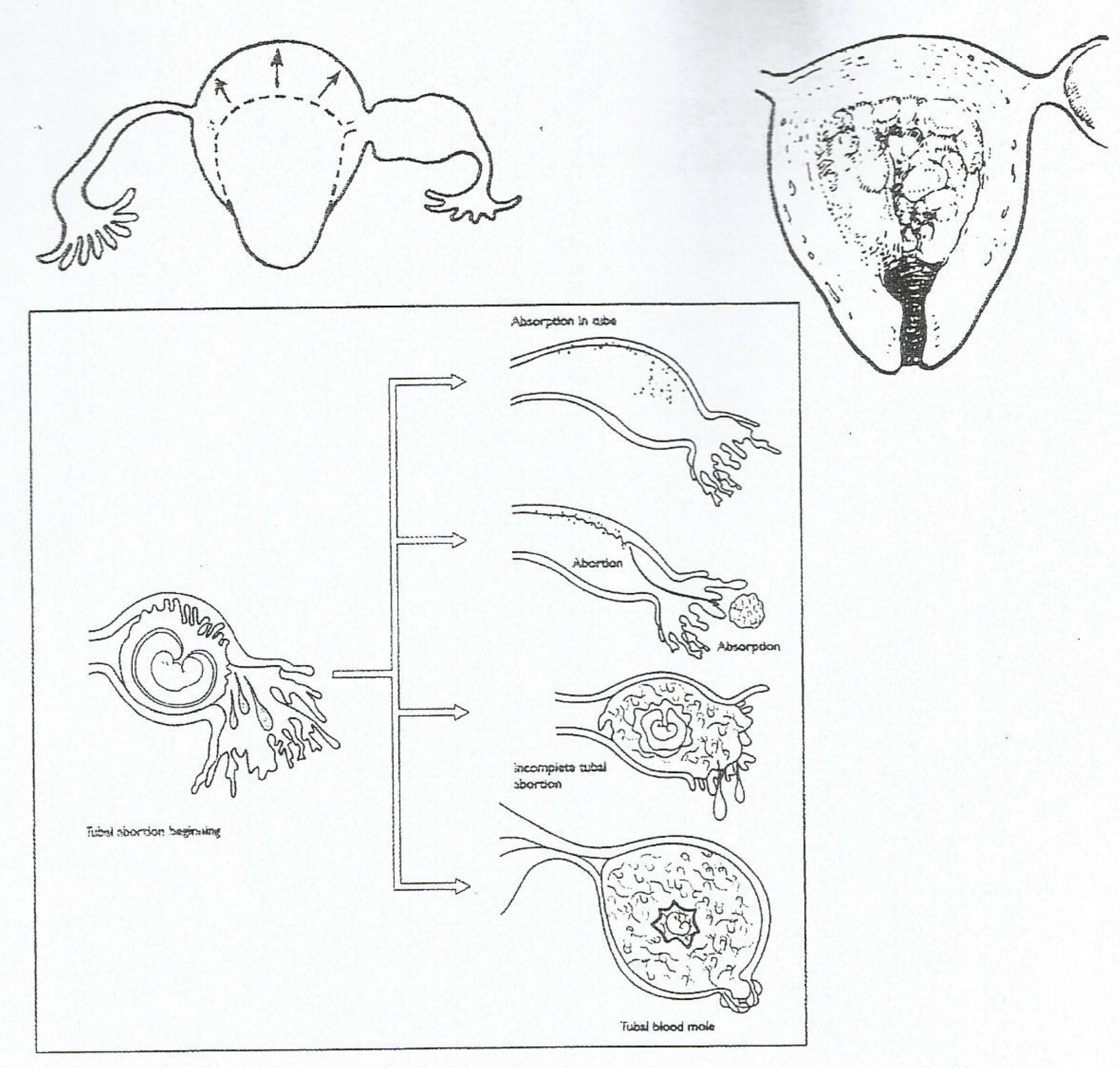
❶ Causes in the tube preventing normal transport (mechanical factors)
adhesions → kinking → time consumption → delayed implantation

- Congenital ⇨ hypoplasia, accessory ostia, diverticula
  → partial obst.
- Traumatic ⇨ surgery on tube.....tuboplasty, tubal ligation
  or surgery near tube.....ovary, uterus, *appendix*
- Inflammatory ✓ ¤ 50% ⇨ PID (**chlamydia** > gonorrhea), *appendicitis*
  PID = STD = chlamydia.
  ↘ peritubal adhesions (esp on the **right** side)
- Neoplastic ⇨ tumors in broad ligament, ovary, uterus (fibroid, ovarian cyst)
  ↘ stretch of the tube & obstruction of ostia
- Miscellaneous
  - Endometriosis → adhesions due to bleeding.
  - ART → ↑ ectopic by 5% (due to
  - Contraception:
    - . POP or Implants → ⇩ tubal motility **(what about COC ¤ ?)** → Protective (anovulation)
    - . IUCD → salpingitis, ⇩ tubal motility (esp if + P), also it can prevent *intra*- but not *extra*-uterine pregnancy

  some say IUCD → ↓ ectopic because it ↓ overall preg. rate.

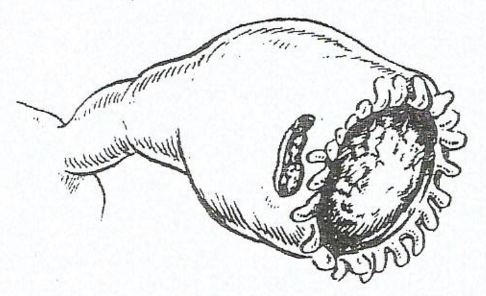
❷ Causes in the fertilized ovum

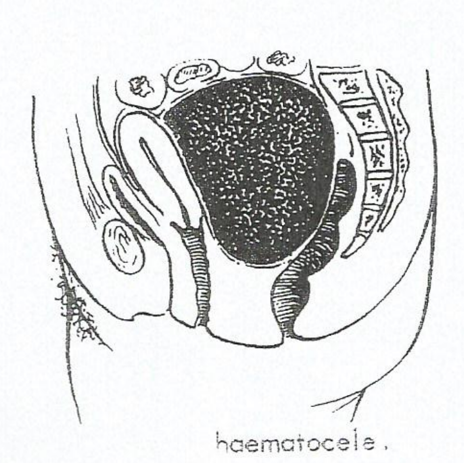
- Early disappearance of zona pellucida
- Early development of trophoblast
- External or internal migration (time consumption) ✗



Sequelae of tubal abortion.

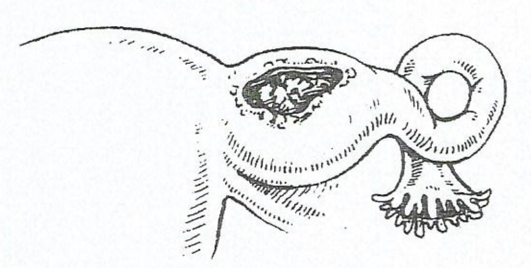
RUPTURE INTO LUMEN OF TUBE (TUBAL ABORTION)

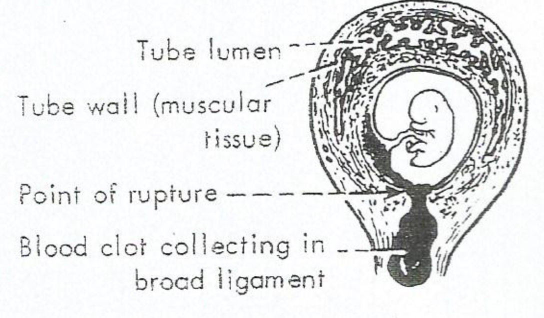




haematocele.

RUPTURE INTO THE PERITONEAL CAVITY





Sometimes rupture is retroperitoneal between the leaves of the broad ligament - broad ligament haematoma.

Pathology

▸ **Tube**

* Any part may be affected (esp ampulla) → enlarged, vascular
* Rarely → *hetero-topic* (intra + extra-uterine)
* Cannot reach > 12 weeks due to early disturbance:
  - Limited tubal distension
  - Poor blood supply & nutrition
  - Thinner decidua → ovum penetrates deep in muscle

▸ **Uterus**

* Symmetrically enlarged (SLIGHT), ↑ed vascularity, hypertrophy
* Decidua (but with no villi)
* ARIAS STELLA REACTION → secretory, proliferative changes with some atypical findings in 10–15% of cases (non-specific)

▸ **Ovary** → one shows CL of pregnancy

...Fates... ...C/P...

↘**Undisturbed**... if diagnosed early < rupture (rare).......undisturbed ect. ❶

↘**Disturbed**........ when the ectopic preg. ruptures:

▸ **Rupture inside the tube:**

- Repeated mild hge around ovum → *tubal mole (hematosalpinx)*
- If hge ⇧ → separation of the fertilized ovum → *tubal abortion*
- Bleeding may be
  - Mild ⇨ *peritubal hematoma*.............subacutely ect. ❷
  - Severe ⇨ *generalized intraperit. hge*....acutely dist ect. ❸
  - Chronic ⇨ *pelvic haematocele*..........chronic dist ect. ❹

▸ **Rupture outside the tube:**

- Due to erosion of the tubal wall → *tubal burst*
- *Earliest* rupture occurs esp. in tubal *isthmus*
- Rupture may be:
  - ☞ ***Intraperitoneal***
    - Mild ⇨ *peritubal hematoma*
    - Severe ⇨ *generalized intraperitoneal hge*
    - Chronic ⇨ *pelvic haematocele*
    - Baby rarely ⇨.....2ry abdominal pregnancy ❺
  - ☞ ***Extraperitoneal***
    - Broad ligamentary hematoma
    - Baby rarely ⇨ 2ry intra-ligamentary pregnancy

Gynacologist / obstetrician must be ectopically minded.
Any Preg. should be considered ectopic till proved otherwise.
" " " " " Twins " " "

Symptom/sign	Ectopic gestation (%)
Abdominal pain	90
Amenorrhoea	80
Adnexal tenderness	80
Abdominal tenderness	80
Vaginal bleeding	70
Adnexal mass	50

Symptoms and signs in ectopic gestation

Asymptomatic → if diagnosed → best chance in TTT

1. Undisturbed ectopic (2%)

Symptoms ......TRIAD......the most imp. is pain......

- ❶ Amenorrhea (short period) + symptoms of early pregnancy
- ❷ Pain → slight dull aching in one iliac fossa (tubal stretch) Commonly right side.
- ❸ Bleeding → usually absent or slight spotting

Signs

- General → signs of pregnancy
- Uterus → soft, slightly enlarged, symmetrical
- Adenexae → . slight tenderness in one fornix → missed for CL of Preg.
  . sometimes a swelling may be palpable (< 3cm)

Early diagnosis needs HIGH LEVEL OF SUSPICION → There is much need for inv.

- History of pdf (e.g. PID, IUCD) + you must be ectopically minded
- May be discovered accidentally during routine U/S of pregnancy

2. Subacutely disturbed ectopic (60%✓✓)

acute abdomen + Int. Hge.

Symptoms

- AMENORRHEA: (may be misdiagnosed c̄ menstrual irregularities)
  - Short period 6 – 8 wks
  - Mostly there is one missed period ✓
- Sudden severe PAIN:
  - Dull aching → tubal distension
  - Sharp stabbing → erosion through the wall + Peritoneum.
  - Colicky → tubal contractions (tubal abortion)
- Vaginal BLEEDING: few days later, very mild.
  - Drop of β-HCG → ↓ E & P → separation of decidua → withdrawal bleeding.
  - Slight dark brown (or rarely as a decidual cast) ↳ Ruptured RBCs.

Signs

- **General**
  - Various degrees of shock → coma in severe cases)
  - ⇧ pulse, ⇩ BP, ⇩ temp., cold clammy skin, oliguria
- **Abdominal**
  - ⇩ed movement of lower abdomen with respiration
  - T, R, RT over lower abdomen (Tenderness, Rigidity, Rebound tenderness)
- **Vaginal**
  - *Cervix* ⇨ extreme tenderness on movement → JUMPING sign
    = cervical motion tenderness ✓✓ → Tenderness of tube.
  - *Uterus* ⇨ difficult to palpate (tenderness) but is slightly enlarged
  - *Adenexae* ⇨ tender enlargement of the affected adnexum + swelling.

hematoma.

The Commonest swelling in D. Pouch is RVF uterus 20%

& RVF uterus vs ch. Pelvic hematocele (ازاي نفرق)

1- Bimanual ex: RVF → uterus is not felt / hematocele → Cx pushed Ant., directed downward

2- PV: RVF → Cx directed forward

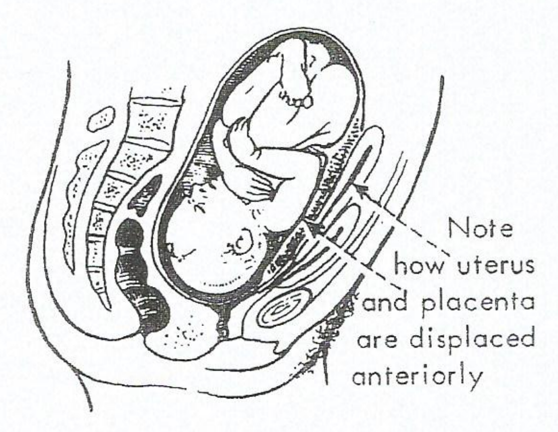
3- Culdocentesis: → hematocele → Blood.

- RVF → Nothing.

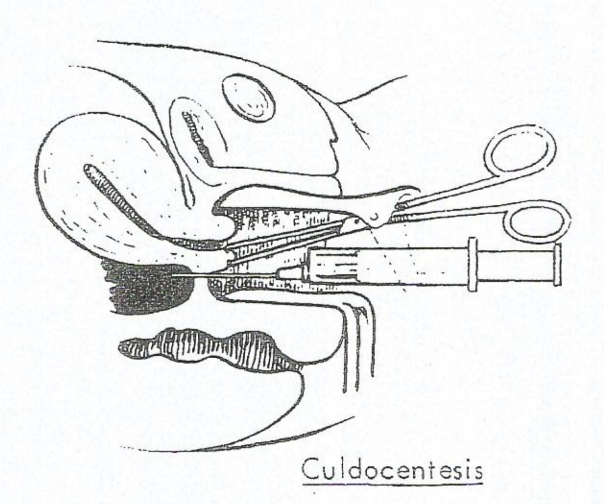
Intraperitoneal haemorrhage and pelvic haematocele

Broad ligament haematoma

Sequelae of tubal rupture.



ABDOMINAL PREGNANCY



Culdocentesis

3. Acute (fulminating) type

Symptoms

.......Short period of **amenorrhea** → sudden severe abdominal **pain**

.......Followed by: massive intraperitoneal hge with shock & **collapse**
± shoulder pain: diaphragmatic irritation by blood

Signs

- **General** → shock (not proportional to external hge)
- **Abdominal**
  - T,R,RT over most of abdomen
  - Shifting dullness ± Cullen's sign
- **Vaginal** → difficult (marked tenderness), but may be easy if......

4. Chronic pelvic haematocoele

Symptoms

.....There is history suggestive of disturbed ectopic preg (the triad)

.....Then blood collects gradually in the D.pouch (most dependent)

.....Leading to pressure symptoms (backache, dysuria, dyschazia, dyspareunia)

Signs

- **General** ⇨ slight pallor ± jaundice ± pyrexia
- **Vaginal** ⇨ tender ill-defined boggy mass in D.pouch pushing cx anteriorly

Treatment......... evacuation by

1- Laparotomy + strong antibiotics
2- Posterior colpotomy (or aspiration guided by TVUS)

5. Advanced abdominal pregnancy

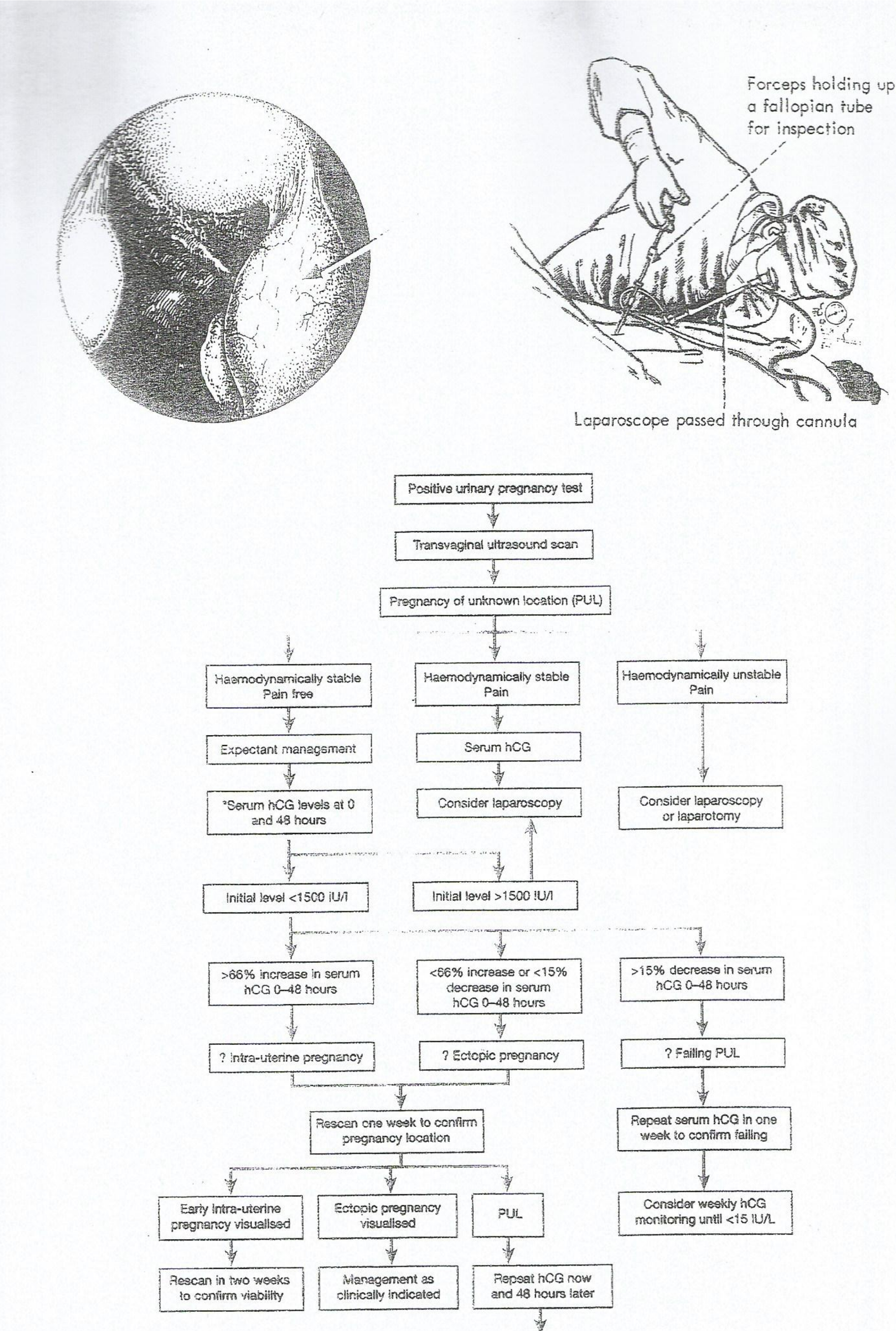
Signs

- *Abdominal* → . Abnormal lie (e.g. high transverse lie)
  . Easy palpation of fetal parts
- *Vaginal* → uterus small & separate from fetus

Investig. ⇨ U/S (CT, MRI):- fetus & placenta are outside uterine cavity

Treatment......... Laparotomy

1- Laparotomy → remove fetus & sac (fetus is malformed in >50%)
2- Regarding placenta:
   - If attached to unimportant structure as omentum → remove it
   - If attached to important structure or great vessels →
     .Cut the cord short & leave placenta for absorption
     .This takes 1-2 years (methotrexate may help absorption)



Algorithm for managing suspected ectopic pregnancy.

U/S: → Cardiac activity detected at 8 w.

Investigations

1] Pregnancy test

- *Serum* β-HCG ✓ (detects 5 mIU/ml) is more sensitive than *urine*
- Intrauterine preg. ⇒ normally doubles / 2–3 days
- Ectopic preg. ⇒ subnormal rise: less than 66% within 2 days
  (But it may be non-viable intrauterine pregnancy)
  ↳ Threatened or missed abortion.

2] Ultrasound — diagnosed on presence of empty uterus / exclusion of uterine preg.

- *Vaginal* U/S is more sensitive than *abdominal* U/S
- Intrauterine preg. ⇒ gestational sac in-utero (5 wks TV.....7 wks TA)
  (But it may be the decidual reaction of ectopic preg)
- Ectopic preg ⇒ a small sac ± fetal echoes outside the uterus
  (But it may be CL cyst of normal preg)

If diagnosis of intraperitoneal hge is evident → proceed directly to laparotomy. Otherwise; in early undisturbed cases → do the following (as diagnosis is difficult/ query)

Hospitalization & follow up of

* Symptoms ⇝ pain
* Signs ⇝ detectable adnexal swelling
* Investigations ⇝ /2 days

❶ Combined U/S + β-HCG)

discrimination zone.

The *discrimination value* at which U/S can detect an intrauterine pregnancy is:- (الرقم الفاصل)

- 6.000 mIU/ml (by abdominal U/S), or
- 2.000 mIU/ml (by vaginal probe)

Any level above this + no intrauterine preg. detected by U/S → will be most probably ectopic

❷ Serial Hb & Hct)

Internal hge is suggested by the progressive drop in Hct in absence of external bleeding

fresh blood → just after bleeding → blood has not yet clotted.

only if - No adhesion - No blood (لو منظار ادخل بمنظار)

If diagnosis is still query → may be misdiagnosed (4-8 %) → small fetus.

* Laparoscopy ⇝ both diagnostic & therapeutic ✓✓
* Culdocentesis ⇝ tapping of blood from D.pouch ✗ (obsolete.)

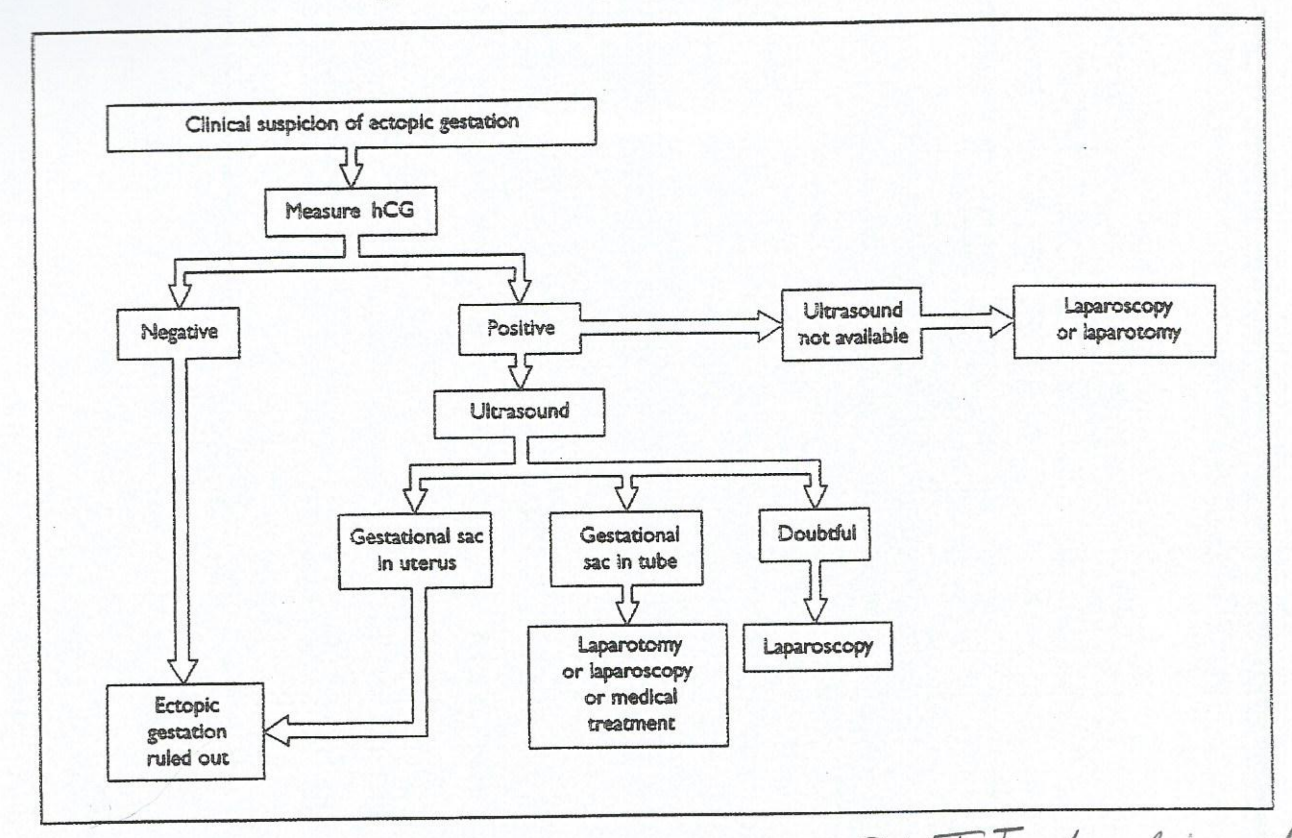
① If blood is clotted → ② V.P. False +ve (blood from B.V.).
→ unclottable blood. due to (After bleeding → clotting in D.Pouch → liquified by fibrinolytic system → No more clotting → Consumption of fibrinogen).

3] Others:

- Progesterone level
  - ≥ 25 ng/ml → normal intrauterine pregnancy
  - ≤ 5 ng /ml → abnormal (*ectopic or non-viable intrauterine preg*)
- D&C ✗✗ ⇒ decidua but no villi (it may disturb an early healthy preg !!)
- EUA ✗✗ ⇒ it may increase disturbance

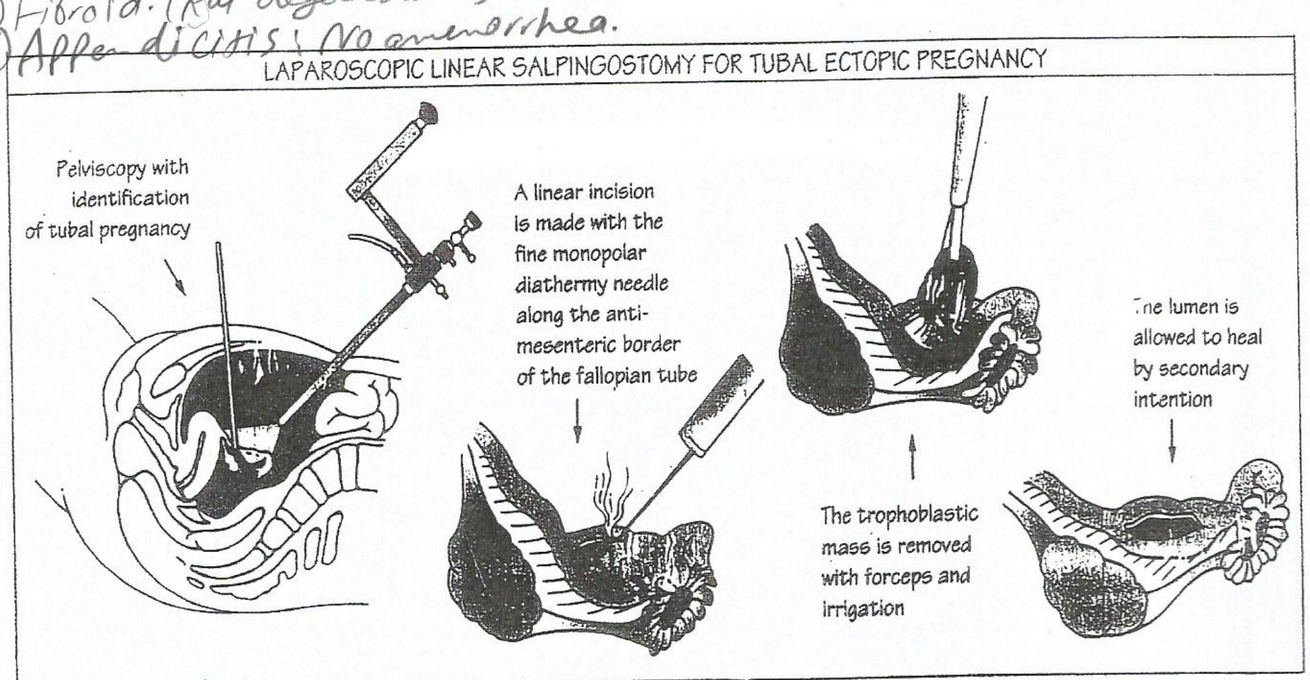
EX. under Anaesthesia.

Inv.: → ① β HCG + TV U/S
→ شوف بعيني: → laparoscope + culdocentesis.
→ عينة دم → Progesterone + Hct
→ obsolete → D and C + EUA.



Algorithm for diagnosis of suspected ectopic gestation.

DD:
① Ectopic.
② PID: usually bilat. + fever + No amenorrhea
③ Persistent CL.
④ Ruptured ovarian cyst: No amenorrhea.
⑤ Fibroid. (Red degeneration).
⑥ Appendicitis: No amenorrhea.
⑦ UTI: Loin pain + dysuria.



⟨ Treatment ⟩

- ▫ Resuscitation ✓✓✓ → anti-shock measures
  ↘ **wide bore cannula + call 4 help**

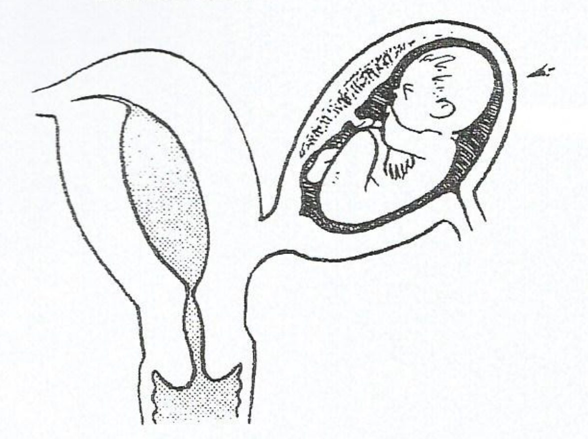
- ▫ Laparotomy (or laparoscopy)
  - Peritoneal toilet → to remove blood
  - 1st inspect the other tube (may be diseased, absent, malformed)
  - **Salpingectomy** → affected tube is removed ✓ (the best; to avoid.....)
    ± . Oophrectomy ✗✗ ➨ to force the other ovary to ovulate monthly
    . No oophrectomy ➨ hormone production
  - **Conservative surgery** if
    ❶ one tube is present or } every attempt
    ❷ mild cases or } should be done
    ❸ low parity } to conserve the tube

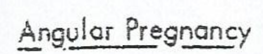
    This is in the form of *linear incision* (at the anti-mesenteric border)
    . Salpigotomy: tube is closed by → sutures
    . Salpingostomy: tube is left open → heal by 2^{ry} intention
    . Partial salpingectomy (never to be done).......high recurrence ✗
    . Milking the tubal contents (esp if near fimbria)...... the worst ✗

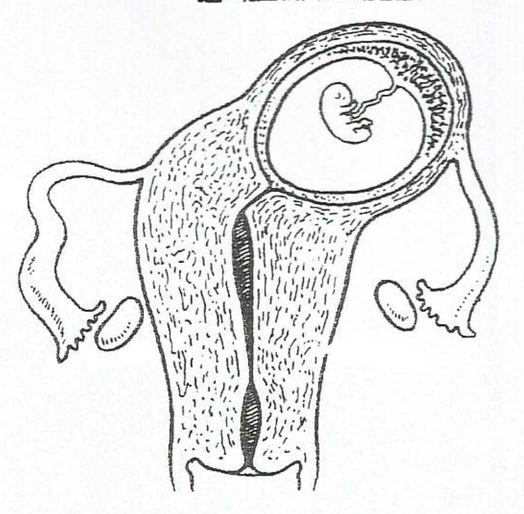
- ▫ Laparoscopy **(diagnostic & therapeutic)**
  - Same procedures as in laparotomy may be done, but needs:
  - Expert team + special equipment + haemodynamic stability (not shocked¤)
  - Adv. → done as a day case

- ▫ Medical ttt **(conservative to fertility)**
  - **Methods** ⇨ . *Methotrexate*✓ folic antagonist (IM 50 mg / m^2)......once
    . *Anti-progesterone* (RU-486).............................Mifepristone
    . *PG-$F_{2\alpha}$* (locally in the sac).......laparoscopic or U/S guided
  - **Criteria**¤ ⇨ (un-disturbed)
    . Sac size < 3 cm, –ve cardiac activity (non-viable)
    . β-HCG < 3000 mIU/ml
    . Patient haemodynamically stable
  - **Follow up** ⇨ . serial Hb & Hct levels, TVUS, β-HCG
    . β-HCG can rise slightly before decline starts (day 4)
    . dose is repeated if no decline by ≥ 15% bet days 4...7
    . surgery is done if no response after 3 doses

- ▫ D&C may be done...........why?
- ▫ If Rh –ve → give anti-D

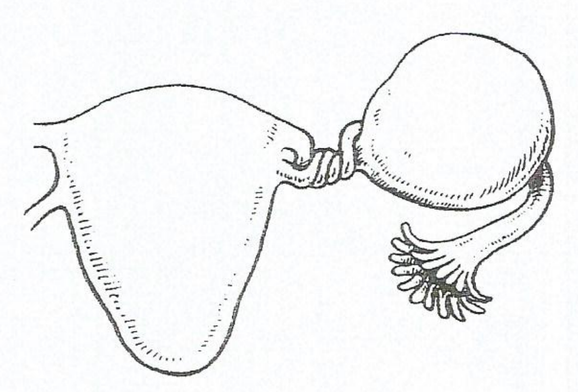






		Abortion	Ectopic
Symptoms	Triad	Am → bl → pain	Am → pain → bl
	- Amenorrhea	Usually present	Short period
	- Pain	Colicky ± backache	Colic, dull, sharp
	- Bleeding	Bright red	Slight dark
Examination	- Shock	Proportional to bl	Not proportional
	- Abdominal	Mild pain	T, R, RT
	- Uterus	= period of amen	Usually < 8 weeks
	- Adenexae	No swelling	Swelling + tender
Decidua + chorionic villi		Present	Absent

imp.



TORSION of PEDICLE of OVARIAN CYST.

……Rare types of ectopic

☆ Ovarian pregnancy

- Usually 2^{ry} to tubal pregnancy
- 1^{ry} is diagnosed by **Spiegelberg** criteria
  - Tube on affected side is healthy
  - Gestational sac occupies position of the ovary
  - Gestational sac is connected to uterus by ovarian ligament
  - Ovarian tissue is found in the wall of the sac

☆ Pregnancy in rudimentary horn

- Usually presents late at 16 – 20 weeks
- It is *medial* to the round ligament while tubal pregnancy is *lateral*
- Treatment → remove horn

☆ Angular (cornual) pregnancy

- At uterine orifice of the tube, late diagnosis (14–16) , more bleeding
- If disturbed → - wedge resection & repair of part of the uterus
  - May need hysterectomy

☆ Cervical pregnancy ⇨ ttt:

- **Hysterectomy** if severe uncontrolled bleeding
- **Conservative measures**:
  1] *Suction evacuation*. To reduce bleeding:
    - Suturing at 3, 9 o'clock
    - Silk suture around the whole cervix (as in cerclage)
    - Balloon tamponade (30 ml) by Foley catheter
    - Bilateral uterine artery embolization by gel-foam

  2] *Methotrexate* local injection in the sac

……DD of ectopic

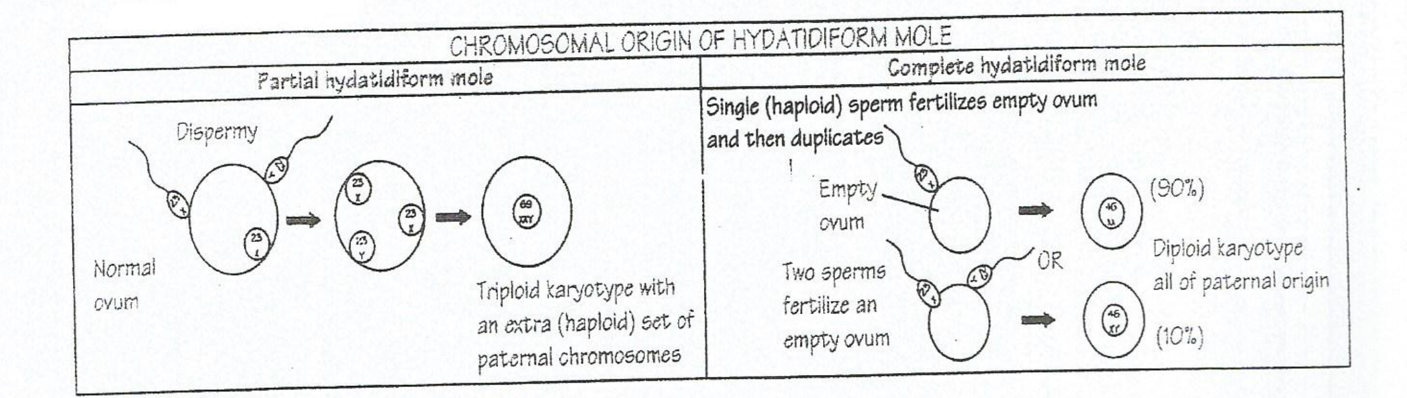
1] Pain from

- ○ *Acute salpingitis* → no amenorrhea, no fainting, fever, pain (usually bilateral), leucocytosis
- ○ *Complicated ovarian mass or fibroid*
- ○ *Acute appendicitis* → no amenorrhea, vomiting, pain usually periumbilical then at Mcburney's point
- ○ *Acute pyelonephritis* → loin pain radiating to the groins with fever & urinary symptoms

2] Bleeding from abortion & vesicular mole

……Later on, after ectopic ⇔

- Contraception → avoid IUCD and POP
- Prognosis → 15%: recurrence ¤, 30% infertility ¤


CHROMOSOMAL ORIGIN OF HYDATIDIFORM MOLE
Partial hydatidiform mole
Complete hydatidiform mole
Dispermy
Normal ovum
Triploid karyotype with an extra (haploid) set of paternal chromosomes
Single (haploid) sperm fertilizes empty ovum and then duplicates
Empty ovum
(90%)
OR
Two sperms fertilize an empty ovum
Diploid karyotype all of paternal origin
(10%)

** Vesicular (Hydatidiform) Mole **

Gestational trophoblastic disease (GTD)		
Benign	Malignant	
Vesicular mole (hydatidiform mole)	*Metastatic*	*Non-metastatic*
	Choriocarcinoma	.Invasive mole .Placental site trophoblastic tumor

Definition

Benign tumor of trophoblast ccc by ⇒ trophoblastic proliferation
+ hydropic degeneration of chorionic villi

Incidence ⇒ commonest in far east ¤ 1/1000

Etiology unknown, m.b.d.t.

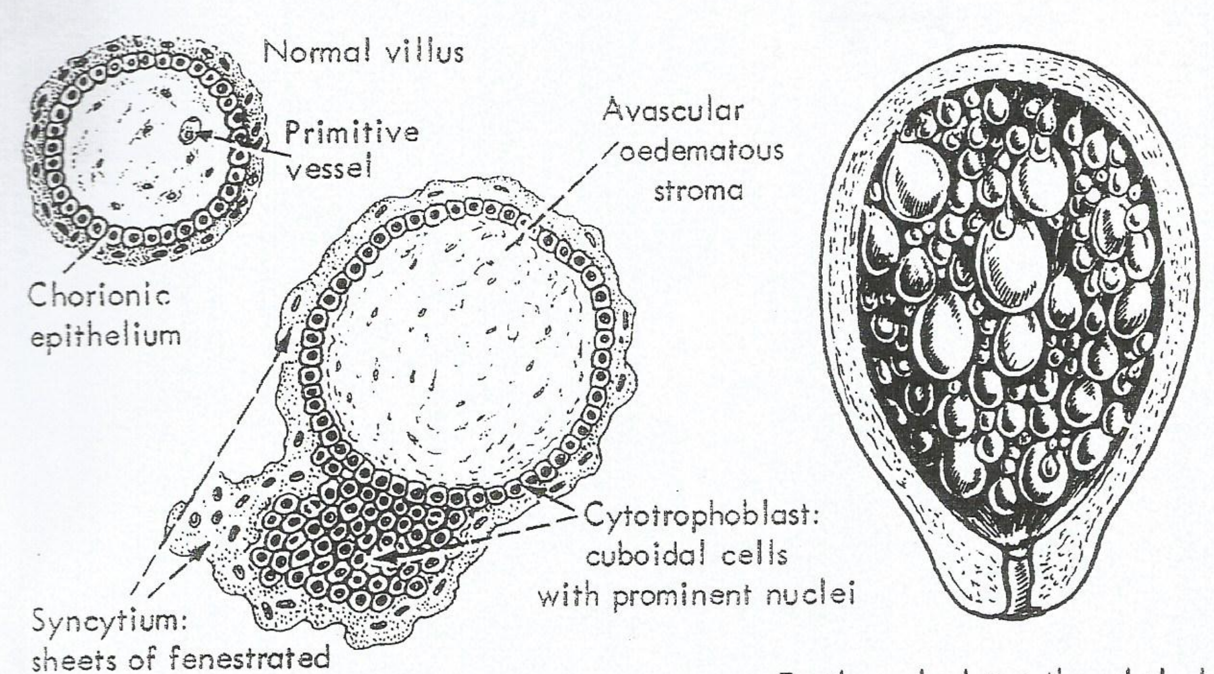
- A primary oocyte abnormality !?
- IMMUNOLOGICAL, GENETIC, →Racial NUTRITIONAL (↓vit A) factors
- Risk factors → previous VM, extremes of age (>35….<20)

Types

1. According to pathology

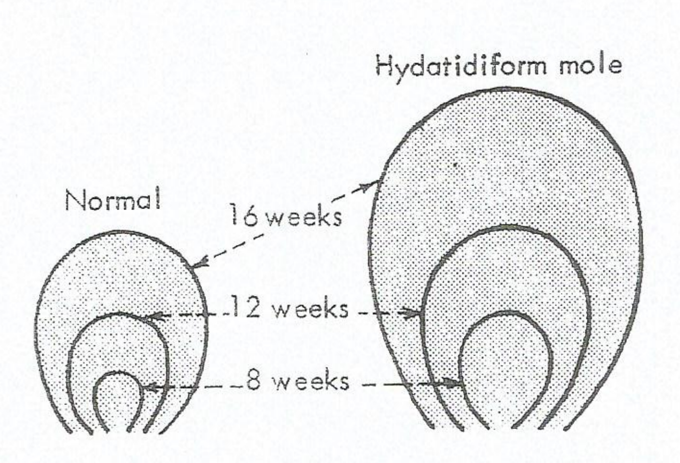
	Complete mole	Partial mole
Incidence	More common	Rare
Malig. change	5–10 %	Rare
Karyotype ¤	46 xx ✓ (all paternal)	Triploid e.g. 69 xxy ✓
Pathology	Only vesicles (no fetus)	Vesicles + fetus → usually aborts in midtrimester
Etiology (Androgenesis¤)	Fertilization of one ovum by [2 sperms or rarely 1 sperm that divides into 2] *followed by* ⤵ ***disappearance*** *of all maternal chromosomes*	A ***normal ovum*** fertilized by 2 sperms or 1 sperm with 46 chromosomes (unreduced genome)

2. According to behavior
   - Benign
   - Invasive mole 15% (choriadenoma destruens) → if perforating the uterus i.e. locally malignant (rarely metastasize)
   - Metastasizing mole (usually metastasizes to lungs ¤) → resolves with ttt



To the naked eye the whole looks like a bunch of grapes.

Types of V.M according to behaviour:
1- Benign V.M.
2- locally invasive V.M. } Benign
3- metastasizing V.M
due to ↑ IUP



Pathology

⇨ Macroscopic

① **Uterus** ⇝ enlarged, studded by vesicles (2 mm to 2 cm) in diameter, each with a small pedicle & contains semitransluscent fluid. No FETUS or PLACENTA

- May be partial or complete
- May affect one twin & not the other

② **Ovary** ⇝ bilateral theca lutein cysts of the ovary -60%- [due to ↑ β.HCG released from the proliferating trophoblast]. They DISAPPEAR SPONTANEOUSLY 2-3 months post-evacuation

⇨ Microscopic

- Trophoblastic proliferation (both cyto– & syncitio– trophoblast)
- Hydropic degeneration of C.T. stroma of villi → vesicles
- No blood vessels (AVSASCULAR PATTERN of VILLI)

Clinical picture

◈ Symptoms

- ***Amenorrhea* +** sympt. of early pregnancy
- ***Uterine bleeding*** (continuous trickling) ±
- ***Pain……but no fetal movements***
  - Dull aching (uterine stretch)
  - Colicky (expulsion)
  - Sharp (perforation)
  - ACUTE ABDOMEN (complicated theca lutein cyst)

◈ Signs

- ***General*** ⇨ ill, anemic / shocked ± signs of comp.
- ***Abdominal***
  - Uterus > period of amenorrhea
  - Uterus doughy in consistency (vesicles with no fetal parts)
  - No fetal parts or FHS (except if ❶ ...., ❷ ....)
  - Bilateral enlarged ovarian swellings
- ***Vaginal*** ⇨ passage of vesicles is *diagnostic* (rare)

Complications ΦΦ

General	Local
PIH <20 weeks	*Haemorrhage , infection*
Hyperemesis gravidarum	*Perforation*
Thyrotoxicosis	*Malignancy* (choriocr.) → % ??
Pulmonary embolism & DIC	*Recurrence* (1-2% ¤)

Criteria of possible development of choriocarcinoma:
1- BHCG: Rising titre, plateau titre, +ve after -ve
2- Irregular bleeding.
3- Hemoptysis → metastasis.

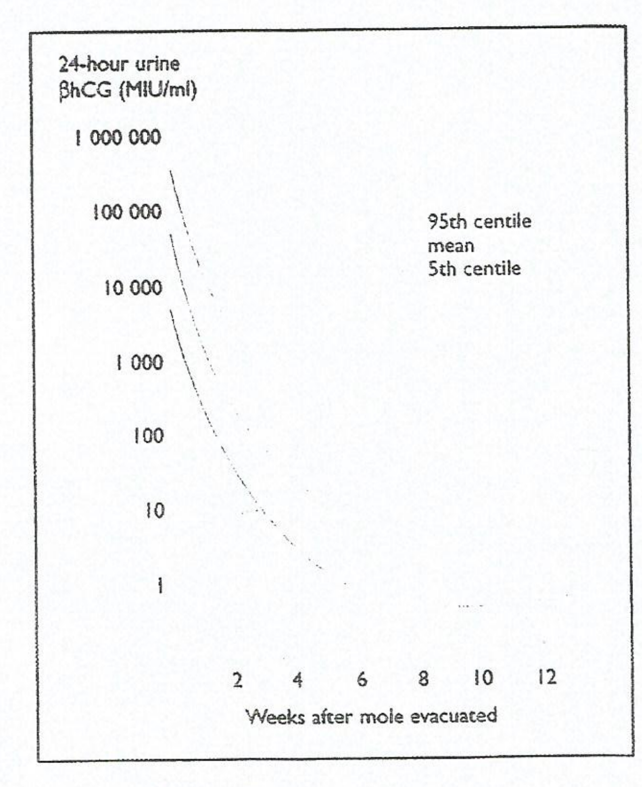
Hysterotomy ✖

* Ensures complete removal *but*
  - Disseminate vesicles
  - Leaves a weak scar
* Previously performed if → severe uncontrollable hge
* Now....it is considered a part of *history* of medicine

Prophylactic chemotherapy

...A single course *methotrexate* (or actinomycin D) may be given at time of evacuation / hysterectomy to
- ⇩ embolisation & metastasis
- ⇩ local uterine invasion

...esp if high risk for recurrence / persistence / metastasis ¤
- Old age (> 40 years)
- β-hCG > 100.000 mIU/ml
- Excessive uterine enlargement
- Theca lutein cysts > 6 cm



Hormone follow-up of benign trophoblastic disease (mean and 95% confidence limits).

Investigations

1- **Ultrasound** ✓ (the best) → SNOW STORM appearance

x-ray+dye ← Amniography → HONEY COMB appearance XX

2- **β-hCG** +ve in high dilutions > 100.000 (more important for *follow up*)

3- **Radiography**: XX Plain X-ray: no fetal skeleton

✓✓ Chest X-ray: for metastasis

Treatment

Resuscitation +

1] Suction evacuation ✓✓ by a wide bore cannula

± curettage to ensure complete evacuation (risk of perforation)

± ecbolics to ⇩ hge (risk of embolism if induction is *started* by ecbolics)

- Don't forget →. anti-D if Rh-ve
  . specimen is sent for histopathology

2] Hysterectomy (in toto)

- In old patients (> 40 years) who have completed their families to ⇩ risk of choriocarcinoma (35% at this age)
- Hysterectomy doesn't prevent metastasis (∴ follow up by β-hCG)
- Theca lutein cysts are not removed surgically *except if complication occur* (e.g. torsion or rupture) or Hge

Follow up

▸ **By β-subunit of HCG** → ⇓ gradually — if no ⇓ → choriocarcinoma; if ⇓ then ↑ again → reccurence

- Every week → till –ve for **3** successive times (<5 mIU /ml)
- Usually becomes –ve within **2–3** months + theca lutein cyst disappear within 2-3 monthes.
- Every month → for **1–2** year/s

▸ **Pregnancy is avoided for 1–2 year/s:**

- To ⇩ recurrence & choriocarcinoma
- COC is used (IUCD ✗ causes irregular bleeding) → as choriocarcinoma causes bleeding.

Any irregular bleeding after (H) of V.m) is considered choriocarcinoma untill proved otherwise.

▸ **Criteria of possible development of choriocarcinoma** → TOP

⟹ **METHOTREXATE**

- β-hCG levels are:
  - *Rising* (doubles in 2 weeks)
  - *Plateau* (failure to ⇩ within 3 weeks)
  - *Returning* +ve after being –ve
- Persistent or recurrent uterine bleeding
- Any evidence of metastasis e.g. chest x-ray
- Biopsy → diagnostic of choriocarcioma



Chapter 3

Antepartum hemorrhage

Vasa Previa

Placenta Previa

Accidental hge

KEY POINTS

1. Nonobstetric causes of antepartum hemorrhage include cervical and vaginal lacerations, hemorrhoids, infections, and neoplasms.
2. Patients typically present with spotting rather than frank bleeding.
3. Nonobstetric causes of antepartum hemorrhage generally require simple management and have good outcomes.
4. Cusco sepeculum examination of the vagina & cervix is very helpful

KEY POINTS

1. Fetal vessel rupture is a rare obstetric complication, usually associated with multiple gestation.
2. It is due primarily to velamentous cord insertion.
3. It is associated with a perinatal mortality of 50%.
4. Patients may present with vaginal bleeding and a sinusoidal FHR pattern.
5. Fetal vessel rupture usually requires an emergency cesarean section.

if local ex (PV) is contraindicated, intercourse is CI.

** Antepartum Haemorrhage **

Definition ⇨ Bleeding from the genital tract *after* 20✓ (28) weeks till *before* delivery of fetus

Etiology

----Maternal----		---Fetal---
Placental site bleeding	**Extra-placental (incidental)**	Vasa previa → The most serious form of APH.
1. Placenta previa (inevitable hge)	1. Local gynecological cause	(the only cause of fetal hge ¤)
2. Placental abruption (accidental hge ✓✓)	2. Excessive show	
	3. Marginal sinus bleeding → diagnosed by exclusion	
	4. Rupture uterus	

……⇨…Vasa Previa …💣…

Definition ⇨ fetal hge due to tear of umbilical vessels running between the presenting part & cervix (The only cause of fetal Hge.)

Incidence ⇨ very rare: 1 /5000 with fetal mortality: 50–75% ¤

Etiology (cong.)
- Velamentous insertion of the cord (defect of warton gel → sagging.)
- Placenta succenturiata , bipartate placenta

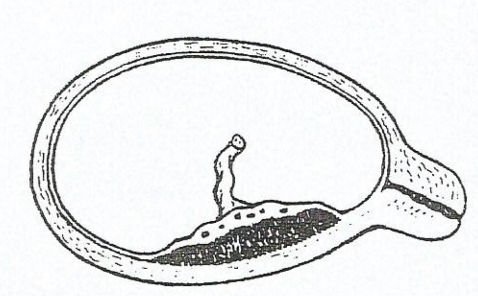
Diagnosis
- **Sympt.** → APhge (*mild* bleeding but *marked* fetal distress)
- **Signs** → vessels are felt as cord like bands crossing amniotic memb. (during labour → Cx dilatation)
- **U/S** → colored Doppler may show the vessels

Investigations

	Fetal blood	Maternal blood
Hb%	16-18 gm%	10-12gm%
Hb electrophoresis	Hb F	Hb A
Blood group & Rh	May differ	May differ
Acid ellution test (Kleihauer Betke test)	RBC's not haemolysed (acid resistant)	Haemolysed (ghost RBC's)
Alkali-denaturation test (Apt test) 0.25% NaOH	No color change → Pink (alkali resistant)	Blood turns → light yellow/brown

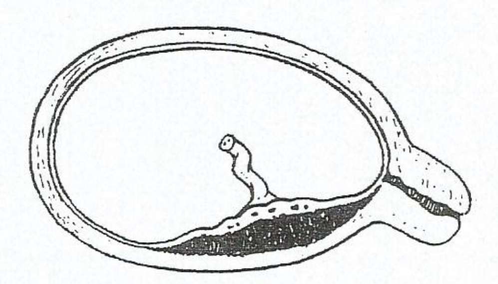
(fetal RBCs > maternal RBCs) (A.P.T (Alkali-Precipitation test).)

Treatment → immediate delivery usually by CS ¤
→ rarely forceps or ventouse if fully dilated



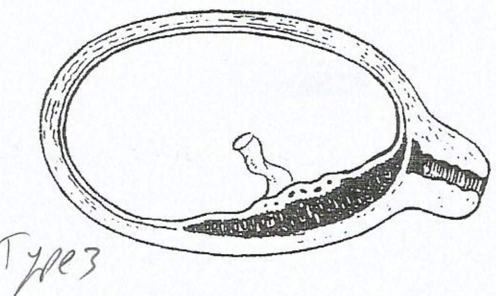
Type 1

The lower margin of the placenta reach the lower segment. ('Low implantation')



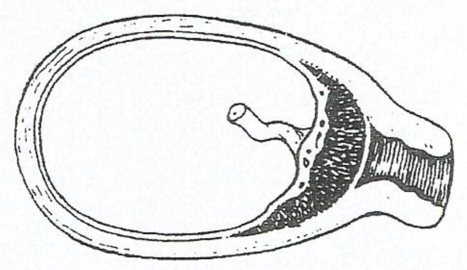
Type 2

The placenta reaches the internal os when closed but does not cover it. ('Marginal').



Type 3

The placenta covers the internal os when closed, but not when fully dilated ('Partial' or 'Incomplete').



Type 4

The placenta covers the os even when the cervix is fully dilated. ('Central' or 'Complete').

Placenta Previa

Definition ⇨ BLEEDING from within the genital tract
AFTER 20 / 28 wks & BEFORE delivery of the fetus
DUE TO <u>*a placenta situated in the lower uterine segment*</u>

Incidence ⇨ 0.5%

↪ More common in <u>MULTIPARA</u>¤ <u>PREVIOUS UTERINE SCAR</u>¤
↪ Recurrence rate…..4–8 %

Etiology

- **Delayed** development of chorion frondosum
- **Delayed** disappearance of zona pellucida
- **Deficient** decidua (↑ parity, ↑ age¤, endometritis) Previous CS.
- **Persistence** of villi in the decidua capsularis

(Basal theory (defected imp. in decidua basalis). Capsular theory.)

* <u>**Large placenta**</u>
  - Twins, D.M., RH → due to hydrops fetalis.
  - Placenta membranacea Cong. anomaly 15-20 cm

Classification depend on time of implantation; more delayed implantation → ↑ degree.

1st °	**PP lateralis** "low lying" ✓✓	60%	Lower margin of the placenta lies in LUS but not reaching the margin of internal os
2nd °	**PP marginalis** "marginal"	30%	Lower margin of the placenta reaches the margin of the internal os
3rd °	**PP centralis incomplete** "partial"	7%	Placenta *partially* covers the internal os
4th °	**PP centralis complete** "total"	3%	Placenta *completely* covers internal os

(Minor 90% — Major 10%)

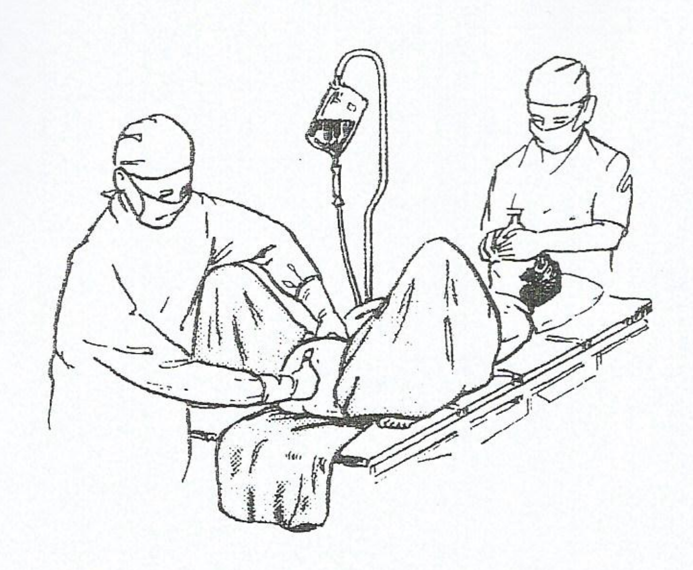
Pathogenesis

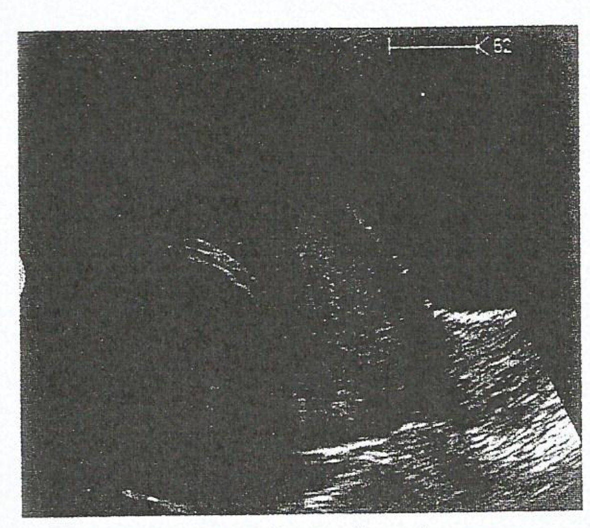
❶ <u>*Pregnancy*</u>:

- Placenta is **inelastic** so bleeding occurs due to stretch of LUS (shearing mech.). Bleeding is augmented by the inability of the weak LUS to compress the torn vessels
- Peak incidence of bleeding is ⇨ 30 – 34 wk¤ → max. period of uterine stretch.
- First bleeding episodes are usually ⇨ mild¤ gradual onset

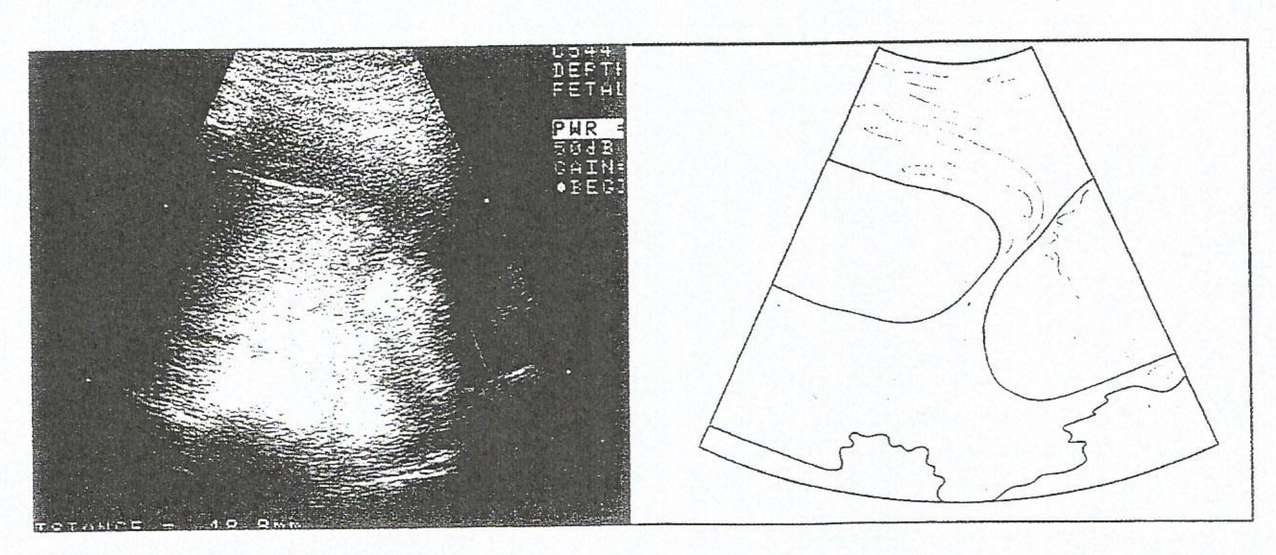
❷ <u>*Labor*</u>: d.t. cx dilatation (rarely may occur for 1st time in labor)

PV is CI if there is bleeding or PROM.
PV is CI after 28 w except after exclusion of P.P.





An anterior placenta praevia extending to just beyond the internal os.



Placenta praevia – type two.

Clinical picture

➡ *Symptoms* ➡ Bleeding:

as LUS is week → - *Fresh bright red* → near vag. (No time for clotting.), Not retained.

No Cont. - *Painless*..............except if…associated with labor pains

due to physiological stretch - *Causeless*............except if…after intercourse or PV

Gradual stretch - *Recurrent*...........except if…placenta is just reaching the LUS

: first presentation during labour.

➡ *Signs*

◈ **General** ➡ pallor or shock (according to degree of bleeding)

◈ **Abdominal** may be higher → Twins, large placenta, DM

- *Palpation* → may be lower → IUGR
  - * Fundal level → corresponds to period of amenorrhea
  - * Umbilical grip → **Lax** uterus, not tender
  - * Pelvic grips → non-engagement ± malpresentations (30%)
- *Auscultation*
  - * FHS are normal except in severe cases (more than ½ placental separation is needed for fetal distress to occur, this is more in cases of ……….)

◈ **Vaginal**

- *Contraindicated* ☠☠ → for fear of placenta P.
- *Except if* ❶ *the patient is in labor &* ❷ *has minor degree*
  - Aim is to determine the possibility of labor
  - In the operative theatre which is ready for immediate interference by CS + available blood
  - This is called a **double set up technique** [2 teams]
  - Placenta (if felt) will be a fleshy tough sponge

Investigations *(ultrasound)*

* The only & best method used (98% sensitivity)

* Repeated serially (every 2 wks) to detect upward migration:

The apparent upward movement of the placenta from the LUS (*due to unequal growth of UUS & LUS*). This may lead to disappearance of p.previa or lessening of its degree

* Thus:-

- P. previa is *more* common at earlier gestational age
- P. previa is *more* likely to persist if diagnosed after 30 wks

* Other methods:- 👎 - Angiography
- MRI → very accurate but expensive
- Thermography → more temp. over placenta

Risk factors for placenta accreta

- Previous retained placenta
- High parity
- Advanced maternal age
- Placenta praevia
- Previous caesarean section
- History of dilatation and curettage or suction termination of pregnancy
- Previous postpartum endometritis

Classification of abnormal placental attachment

Type	Incidence	Pathology
Placenta accreta	75–78%	Invades superficially into the myometrium
Placenta increta	17%	Invades deeply into the myometrium
Placenta percreta	5–7%	Invades through the myometrium and penetrates the outer serosal layer of the uterus. It may invade adjacent structures, including bladder and bowel

Complications ΦΦ

➡ Maternal

◈ **Pregnancy** ➡ APhge (anemia if mild, shock if severe)

◈ Labor

1st stage

- Prolonged labor (due to hge & uterine inertia)
- PROM (presenting part not well fitted on the cervix) ⊊ cord prolapse & infection

2nd stage: difficult (obstructed labor + malpresentations)

3rd stage: PPHge

- *Atonic* (poor contraction of LUS + poor maternal condition)
- *Traumatic* → friable lower uterine segment.
- *Retained placenta* (5%) → placenta accreta (d.t. poor decidual development) → incidence ↑ with no of CS ¤

..........(APhge predispose to PPhge).........

..........(APhge weakens, PPhge kills).........

◈ Puerperium ...S³

Sepsis

- Poor general condition (shock + exhaustion)
- Placenta (friable + near vagina + retained parts)
- Premature rupture of membranes
- Increased surgical interference

Secondary postpartum hge (retained placental parts).

Subinvolution of uterus

➡ Fetal

◈ Preterm labor............................PTL } due to
◈ Intrauterine growth retardation ......IUGR } poor
◈ Congenital fetal malformation.........CFMF } blood
◈ Intrauterine fetal death.................IUFD } supply

Maternal mortality (<1%)	**Perinatal mortality** (5%)
- Hemorrhage - Sepsis	- All complications - Especially → { Prematurity }

Treatment

1] **Conservative ✓✓ if** ➡ . Bleeding → mild
. Fetus → not mature, not distressed
. Mother → not in labor

☆ *Hospitalization…aim*: keep condition under control till maturity

1.Mother :
- Bed rest, no P/V, no vaginal douching
- Correct anemia by diet, iron ± blood transfusion.
- Close observation, ready blood stores

2.Fetus :
- Give steroids for lung maturity
- Serial tests for fetal well being

3.Placenta : follow up placental migration by U/S

2] **Termination if** ➡ . Bleeding → severe........or
. Fetus → mature or distressed ....or
. Mother → spontaneous onset of labor

☆ *Anti-shock measures ✓✓*

☆ *Birth either*

C.section ✓✓: ⊶ + severe bleeding, 2nd posterior, 3rd, 4th

➡ LSCS → . better control of bleeding (near placental bed)
. leaves a strong scar

- If placenta is found anterior
  - Incise the placenta & deliver baby through it ✓
  - Reach round it till head is felt
- Control of placental bed hemorrhage
  - Ecbolics + massage + hot packs
  - Under-running sutures
  - Uterine artery ± bilateral internal artery ligation
  - Supra-vaginal hysterectomy

Vaginal : ⊶ + mild bleeding, 1st & 2nd anterior (why?)

➡ AROM ± oxytocin

- *1st stage* → continuous fetal & maternal monitoring
- *2nd stage* → no forceps or breech extraction
- *3rd stage* → guard against postpartum hge

☆ *Care of the maternal complications.........shock, PPhge*

☆ *Care of the newborn*

Abruptio placenta (accidental hge)

Definition ⇨ BLEEDING from the genital tract of placental site origin
AFTER 20 / 28 wks & BEFORE delivery of the fetus
DUE TO separation of a normally situated placenta

Incidence ⇨ 1 / 200–500 [> in **PG** with PIH, **elderly PG, GMP**]

Etiology Φ ……………………idiopathic in many cases……………………

1] PDF : .↑ age, parity…..smoking, alcohol
.↓ folic acid ¤, vit.C., vit.K
. Previous accidental hge

2] Maternal disease :

- Preeclampsia (the most important → 50%) ✓✓
- Vascular wall defect (DM., collagen disease)

3] Trauma to the abdomen

- Accident.
- External Cephalic Version (esp if traction on short cord)

4] Sudden ⇩ in intrauterine pressure:

- After sudden ROM in polyhdraminos
- After delivery of first twin.

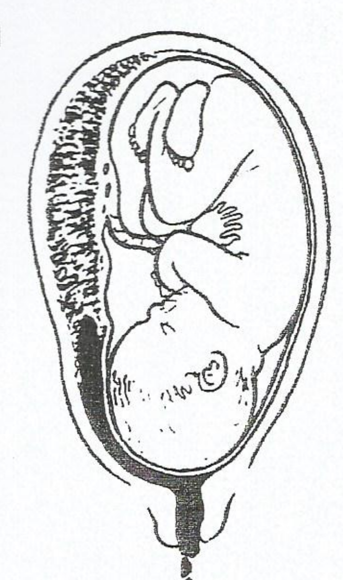
5] Placental abnormalities :

- Circumvallate placenta
- Placenta implanted on *scar, septum, fibroid*

Pathogenesis

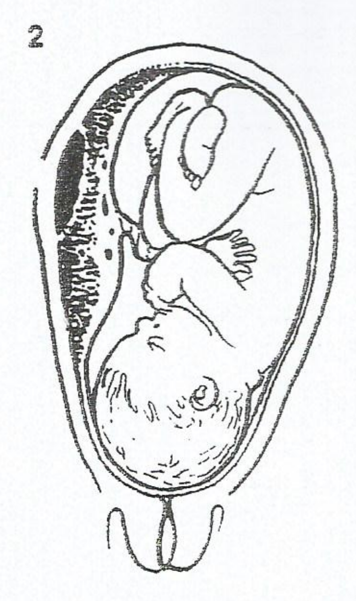
- Injury of vessels in the choriodecidual space → retroplacental hematoma → automatic extension due to rupture of more vessels by the collecting hematoma → more expansion of the hematoma
- Blood may escape between muscle fibres of myometrium (Couvelaire uterus / uteroplacental apoplexy) → black flabby uterus incapable to contract
- Extension of blood may produce ecchymoses below peritoneum or even *rupture* uterus & internal hemorrhage
- Release of tissue *thromboplastin* + *consumption* of the clotting factors within the hematoma → will lead to DIC → FDP's:- (renal failure + tocolytic effect → atonic PPHge)

1

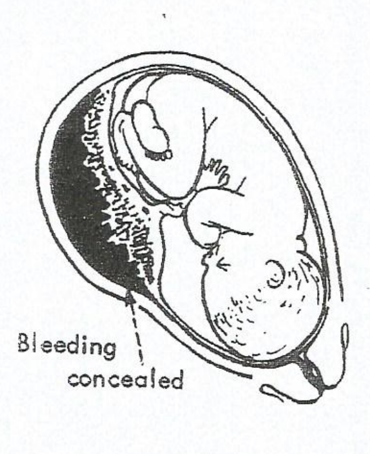


External bleeding alone causes little upset.

2



3



Comparison of the clinical picture in the various grades of accidental haemorrhage

	Severity of bleeding		
	Mild	**Moderate**	**Severe**
Pulse	No change	Raised	Raised
Blood pressure	No change	Lowered	Lowered
Shock	None	Often	Always
Oliguria	Rare	Occasionally	Common
Hypofibrinogenaemia	Rare	Occasionally	Common
Uterus	Normal	Tender	Tender and tense
Fetus	Alive	Usually dead	Dead
Blood loss (litres)	<1	1–3	3–6

Types

1] According to bleeding

1. Concealed (10%)	The blood separates part of the placenta but does not reach the vagina
2. Revealed (30%)	The lower margin of the placenta separates and the blood tracks down wards (between the membranes & uterus) to escape through the vagina.
3. Mixed (60%) ✓	Commonly starts concealed then become revealed

2] According to severity

		C/P	Fetus	Shock	DIC
Class 0	Mildest	-ve	Alive	-ve	-ve
Class I	Mild	+ve	Alive	-ve	-ve
Class II	Moderate	++ve	Distressed	+ve	-ve
Class III	Severe	+++ve	Dead	++ve	+ve

Clinical picture

1] **Symptoms**

- Pain ✓ (SUDDEN, SEVERE, CONTINUOUS abdominal pain)
- Bleeding.....dark, clotted (absent in concealed type)
- Shock (hypovloemic + neurogenic in concealed)

2] **Signs**

▸ ***General***

- Signs of etiology e.g. PIH (but arterial blood pressure may be apparently normal i.e. hypotension due to shock is masked by PIH (decapitated B.pr.) ∴ Hypovolemia is better detected by CVP
- Shock [may not correspond to the external bleeding]
- Signs of complications e.g. DIC

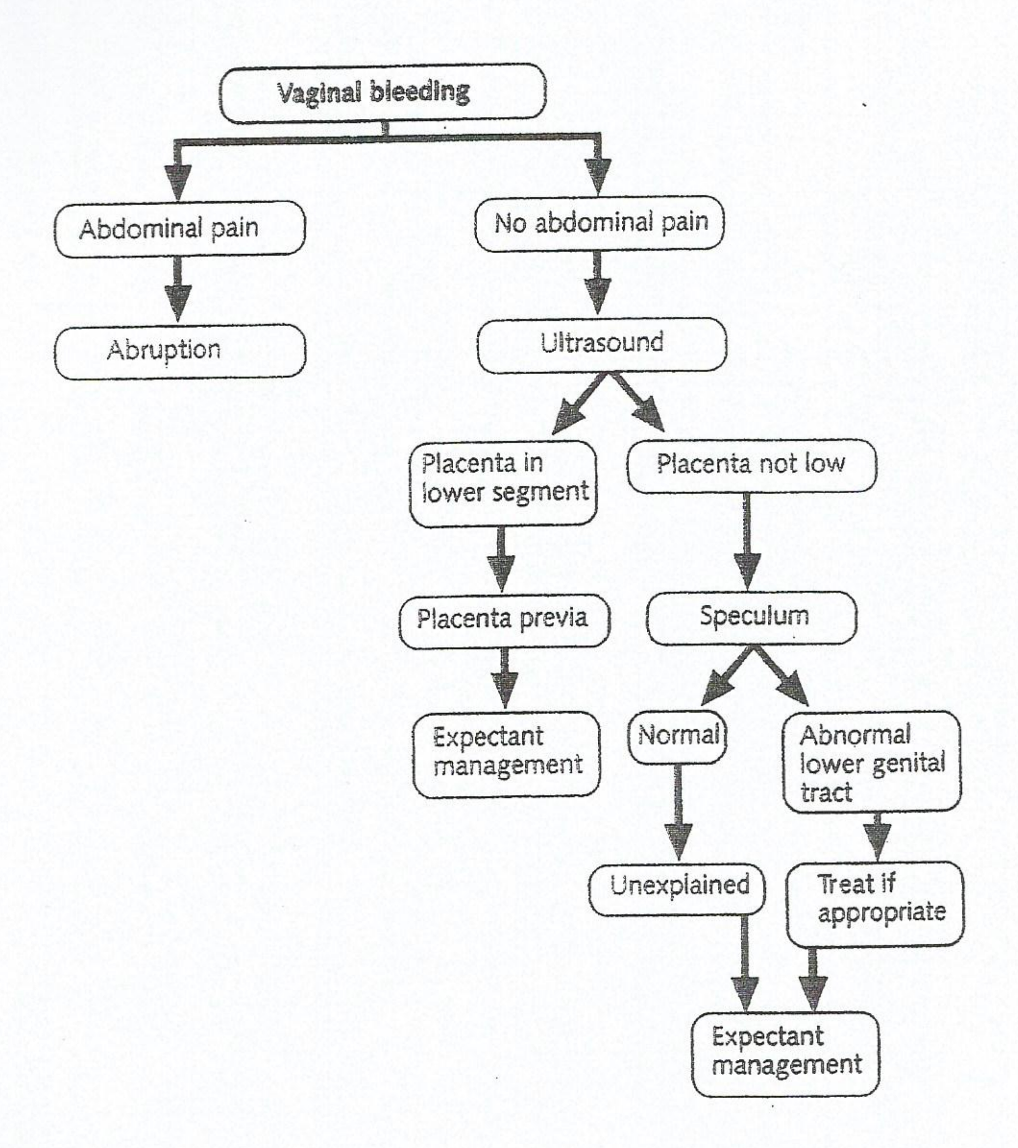
▸ ***Abdominal***

- Palpation :
  - * Fundal level → higher than period of amenorrhea
  - * Umbilical grip → ↑ basal uterine tone *(board like rigidity)*
  - * Pelvic grip → normal presentation + engaged head
- Auscultation : according to severity (*distressed* ✓ or absent)

▸ ***Vaginal***

- Contraindicated (*No PV in any case of APHge*)
- Only done after exclusion of P.previa by U/S ⇝ well engaged head & very tense membranes (if ruptured → bloody liquor)

Algorithm for APH.



Investigations

1. *Etiology* → preeclampsia
2. *Diagnosis* (U/S) → . exclude placenta previa ✓
   . may find retroplacental hematoma
3. *Complication* → DIC, renal function tests

Differential diagnosis

1. Causes of *acute abdomen* in late pregnancy (concealed or mixed)
2. Causes of *antepartum haemorrhage* (revealed or mixed)

	Mixed accidental hge.	**Placenta Previa**
History **- bleeding**	- Once - has etiology - painful - dark clots	- recurrent - causeless - painless - fresh blood
Examination **- General**	* Etiology e.g. → PIH * Shock > hge. * DIC	* no etiology * shock = hge.
- Abdominal	Tender, hard	Painless, soft
- Vaginal	- no placenta - cephalic - well engaged	- placenta felt - malpresentation - not engaged
U/S	normally situated	in LUS

Complications

Φ ✍

➯ **MATERNAL** [MMR......... 1%] ¤

♠ *Due to the developing hematoma*

1] Shock

. Due to.......APhge, PPhge (atonic✓ ± traumatic ± DIC)
. Leading to - Renal failure (shock + PET + DIC)
- Sheehan syndrome

2] DIC → accidental hge is the commonest cause of DIC

♠ *Due to the high intrauterine pressure*

3] Couvelaire uterus (utero-placental apoplexy) &
Rupture uterus → intra-peritoneal haemorrhage

4] Amniotic fluid embolism

♠ *Due to the etiology* e.g. complications of preeclampsia

➯ **FETAL:** PTL ✓........ IUFD ✓...... IUGR.....(why?)

History

Alexandre Couvelaire (1873–1948) was the first to describe extensive haemorrhage into the myometrium; he recognized that it was impairing the myometrium's ability to contract, such that in the case he reported, a caesarean hysterectomy was required. He also was an early proponent of caesarean section for placenta praevia.



Couvelaire uterus

KEY POINTS

1. Placental abruption accounts for 30% of all third-trimester hemorrhages.
2. Fetal mortality rate can be as high as 35%.
3. Patients usually present with vaginal bleeding, painful contractions, and a firm, tender uterus; 20% of cases present with no bleeding (concealed hemorrhages).
4. Major risk factors include hypertension (chronic or gestational) and previous history of abruption.
5. Placental abruption can be complicated by hypovolemic shock, DIC, and preterm delivery.
6. Patients can be delivered vaginally if they are stable; cesarean delivery is necessary in the unstable patient or when fetal testing is nonreassuring.
7. Risk of recurrence increases in subsequent pregnancies.

Treatment

1] <u>Termination</u> ✓is the usual fate, as:-

- *Bleeding* is usually → severe
- *Fetus* is usually → distressed
- *Mother* usually enters in → spontaneous labor

a] <u>Anti-shock measures</u> ✓✓
b] <u>Birth by</u>:

1- Cesarean section ✓✓: ⊶ +

- Maternal <u>or</u> fetal **distress** <u>&</u> delivery is **not** expected soon
- MOST IMPORTANT → coagulation defects should be corrected **first**
- Hysterectomy → in severe atony or Couvelaire ¤ *(if failed to contract)*

2- Vaginal if : ⊶ +

- Especially if the fetus is **dead** <u>or</u> the patient is **advanced** in labor
- Usually *easy* (well engaged head) & *rapid* ($\uparrow^{ed}$ basal uterine tone)
- Early AROM (why..........3**R**) ± oxytocin
  - ➡ **R**elieve.......the high IUPr
  - ➡ **R**eveal........any internal hge.
  - ➡ **R**elease......PG...accelerate labor

1st stage → continuous monitoring (F & M)
2nd stage → usually rapid
3rd stage → guard against PPhge

c] <u>Treatment of *etiology*</u> (PET) & <u>*complications*</u> (DIC)
d] <u>Care of the newborn</u>

2] <u>Conservative</u>, rare

- *Very rare* (chronic abruption) : as once abruption occurs, there will be → automatic extension → severity → complications
- *Indications*:
  1. Bleeding → mild (usually revealed or small retroplacental hematoma).
  2. Fetus → not mature < 37 wks
  3. Mother → not in labor
- *Aim to* → control the condition till fetal maturity.
  - Mother:
  - Fetus:
  - Placenta: serial U/S to follow size of <u>*hematoma*</u>
- *Continue* → till *any* of the indications of termination occur

Chapter 4

Postpartum Hemorrhage

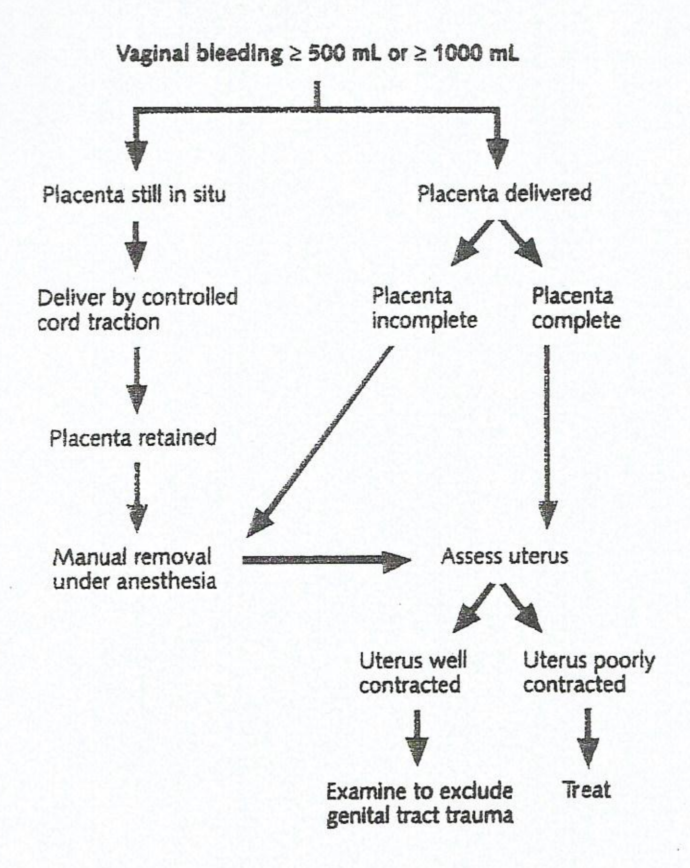
Atonic

Traumatic

Retained Placenta

DIC

Acute Inversion



Algorithm for the management of early PPH.

Causes of PPH

- Tone: uterine atony
- Tissue: retained products of conception
- Trauma: genital tract laceration
- Thrombin: clotting abnormalities

Postpartum Haemorrhage

Definition

- It is haemorrhage from the genital tract
  AFTER delivery of the fetus
  TILL the end of puerperium EITHER
    - To a degree affecting maternal general condition
    - More than 500 cc
    - Causing haematocrit drop > 10 %
- Incidence has been reduced from 15 → 5% (d.t.↑ use of ecbolics)
- It is the commonest cause of MMR in *developing* countries (30%)
- More common with history of previous PPHge ¤

Types

Primary PPhge. (hge within 24 hours of delivery)

- ***T**one*..............Atonic.........................(90% of causes)
- ***T**rauma*.........Traumatic.....................(extra-placental site hge)
- ***T**issue*............Retained placenta.........(3rd stage hge)
- ***T**hrombosis*....Coagulation defects..........(mostly DIC)
- Acute inversion of the uterus...............(v.v.rare)

Secondary PPhge (hge after 1st day till end of puerp.) Φ Φ

1- Etiology

a- The **commonest**..........retained fragments of placenta ± infection
b- The most **serious**.......choriocarcinoma
c- **S**epsis...................separation of a slough → bleeding
d- **S**ubinvolution..........inversion of the uterus
e- **S**ubmucous polyp.....if ulcerated
f- Others
  - **Local** gynecological disease → cervical ulcer
  - **General** → coagulation defect

2- Assessment

* History (mode of delivery)
* Examination (general , abd , local: cusco)
* Investigation (U/S , β-HCG)

3- ttt of the cause

* Retained fragments.........
  - if small remnant / mild bleeding → methergine + antibiotics
  - if large remnants / severe bleeding → evacuation guided by U/S
* Choriocarcinoma.......... chemotherapy

① Atonic PPhge 

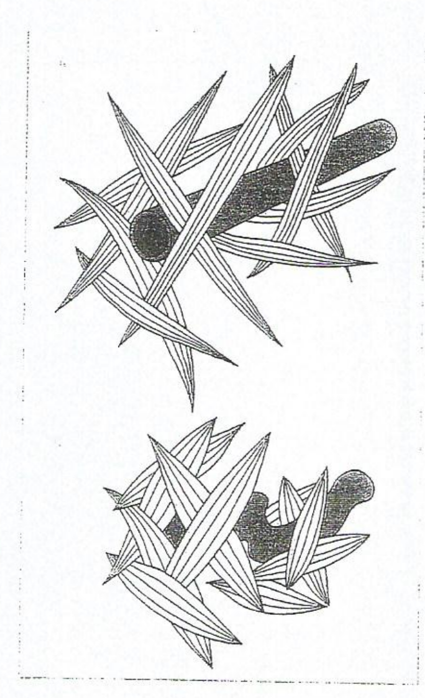
Definition

- Postpartum bleeding due to weak contractility & retractivity
- Constitutes about → 90% of cases (the commonest ¤)

Etiology Φ

[1] During pregnancy

- APhge (pl. previa , accidental hge)
- Maternal disease (anemia , pre-eclampsia)
- Over-distended uterus (twins , polyhydramnios)
- Long use of tocolytics

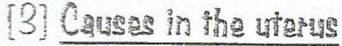


[2] During labor

↪ 1st Stage ⇛
- Prolonged 1st stage
- Excessive straining
- Overuse of sedatives
- Chorioamnionitis
- Full bladder / rectum

↪ 2nd Stage ⇛
- Prolonged 2nd stage
- Excessive manipulation
- Deep anesth. esp. halothane
- Precipitate labor

↪ 3rd Stage ⇛ Retained parts of placenta

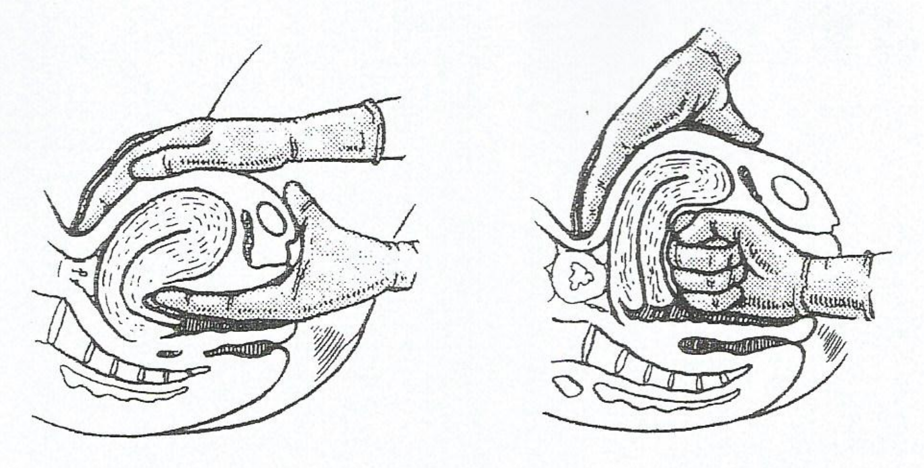


[3] Causes in the uterus

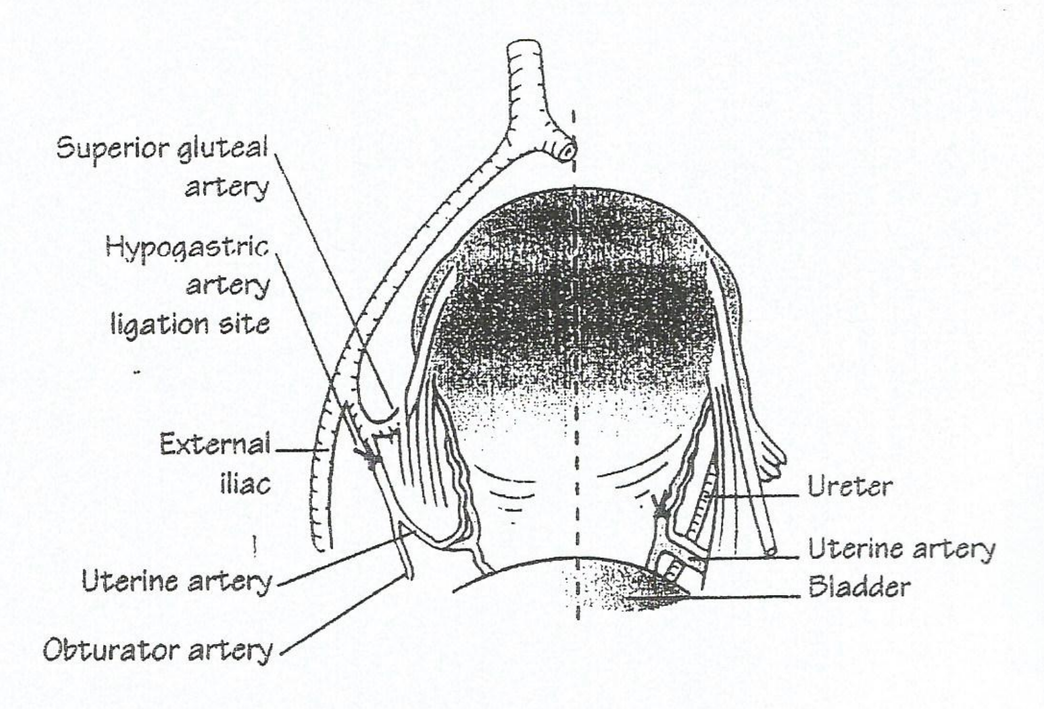
- Multiple fibroids
- Congenital malformations
- Grandmultipara

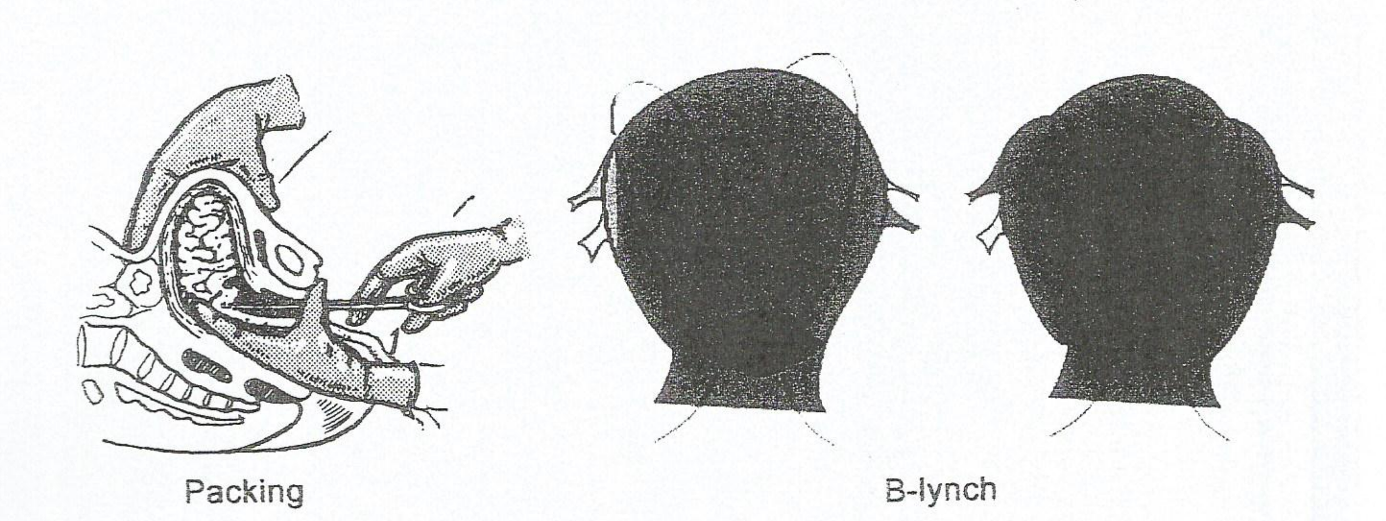
Clinical Picture

- **History** → severe vaginal bleeding after delivery of the fetus & placenta
- **Examination**
  - *General* → Shock
  - *Abdominal* → Uterus soft & enlarged
  - *Vaginal* → to exclude traumatic PPhge.



Bimanual Compression





Packing

B-lynch

Treatment

☆ **Prophylactic**

- Avoid all pdf. → proper ANC
- Proper management of 1st, 2nd & 3rd stages of labor

☆ **Active:** **Resuscitation** +

1st line ⇨ - *massage* ¤

- Ecbolics (oxytocin, methergine, PGE_1 – misoprostone 800μg –)
- Empty bladder + stop halothane

2nd line ⇨ *exploration of birth tract under G.A:*

- Exclude trauma
- Evacuate blood clots or retained placental parts

3rd line ⇨ *bimanual compression of the uterus*

- Closed fist of the right hand is placed into anterior fornix
- Left hand is placed abdominally to compress the uterus inbetween → kink uterine vessels & compress placental site
- This is continued with the aid of assistant every 15 min.

4th line ⇨ *laparotomy*

➤ Pt. completed her family → Supravaginal hysterectomy

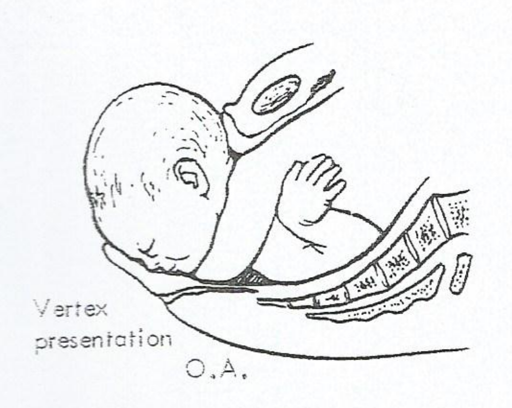
➤ Pt. not completed her family →

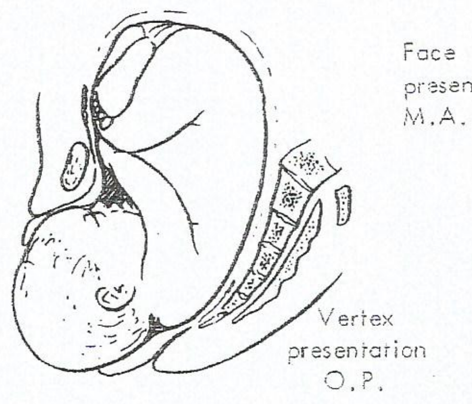
- Direct uterine massage, hot fomentations
- Intra-myometrial prostaglandins ¤ ($PG\text{-}F_{2\alpha}$)
- Bilateral uterine & ovarian artery ligation
- Bilateral internal iliac a. ligation
  - ◈ Difficult surgical technique, with possible injury to ureter, IIV
  - ◈ Uterine blood supply will then depend on collaterals
- If all failed → Supravaginal hysterectomy

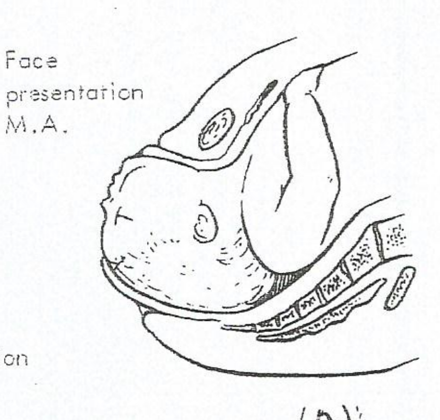
Recently → . B-lynch operation (Brace suture)
. Balloon (hydrostatic intrauterine temponade)
. Bilateral uterine a. embolization using polyvinyl-alcohol particles (gelfoam)

Previously → methods not done now ✗✗

* Uterine douche by Bozeman double way catheter using warm saline or antiseptic → stimulate contraction, remove remnants.
* Uterovaginal pack for 24 hrs (Abcs + ecbolic + catheter) → stimulate contraction, pressure over bleeding site.

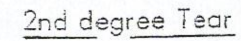






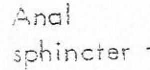
<u>1st degree Perineal Tear</u>

Vaginal and perineal skin are torn, but the perineal muscles are intact.



<u>2nd degree Tear</u>

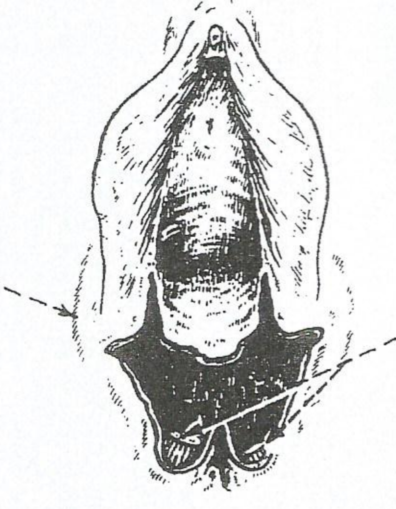
The perineal body is torn right down to (and sometimes partly involving) the anal sphincter. The vaginal tears often extend up both sides of the vagina.

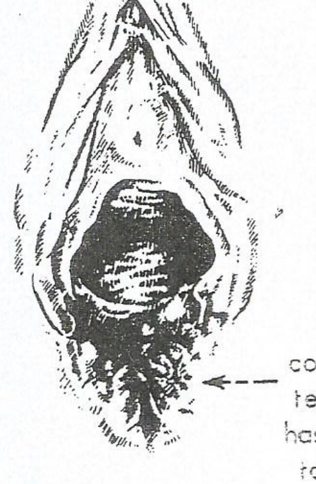


<u>3rd degree Tear</u> - "<u>Complete Tear</u>"

The whole anal sphincter is torn apart, and there may be a tear of the rectal wall. Note how the ends of the sphincter muscles tend to retract.

This injury, if not repaired, leaves the patient with faecal incontinence.





② Traumatic PPhge



[1] Perineal tears

Etiology: Φ

1] *Over stretch of perineum*
- If head is allowed to extend before crowning
- Malpresentation : Face , DOP
- Large head
- Narrow vaginal introitus or subpubic arch

2] *Rapid stretch of perineum*
- Precipitate labor
- After-coming head of breech.

3] *Causes in perineum*
- Rigidity (e.g. elderly PG or previous scar)
- Edema (e.g. PET or obstructed labor)

4] *Injury of perineum* ⇨ forceps…ventouse….destructive operations

Degrees (incomplete or complete)

1st degree → vaginal wall + perineal skin
2nd degree → + perineal muscles ± levator ani
3rd degree → + external anal sphincter
4th degree → + rectal mucosa (some consider it 3rd degree)

* *Hidden perineal tear*
Tear in the perineal muscle without any visible tear of the vagina or skin → predisposes later on to rectocele

Complications

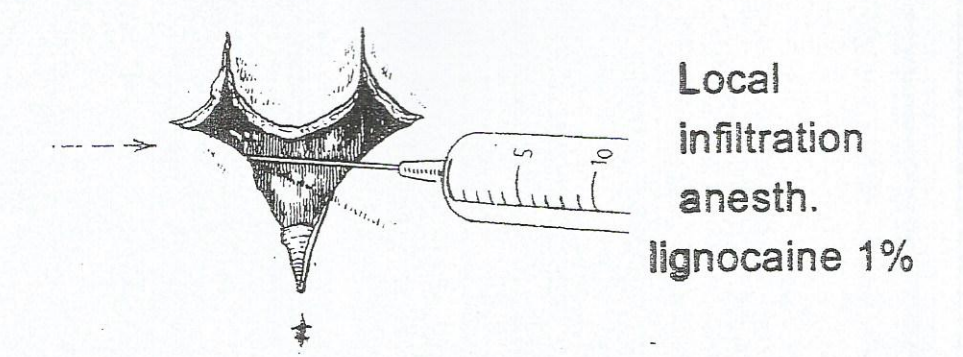
Early → Hge ± infection
Late
- incomplete tear (1+2) → prolapse
- complete tear (3+4) → incontinence of flatus & stools
- improper healing → recto-vaginal fistula
- poor healing → scar →dyspareunia

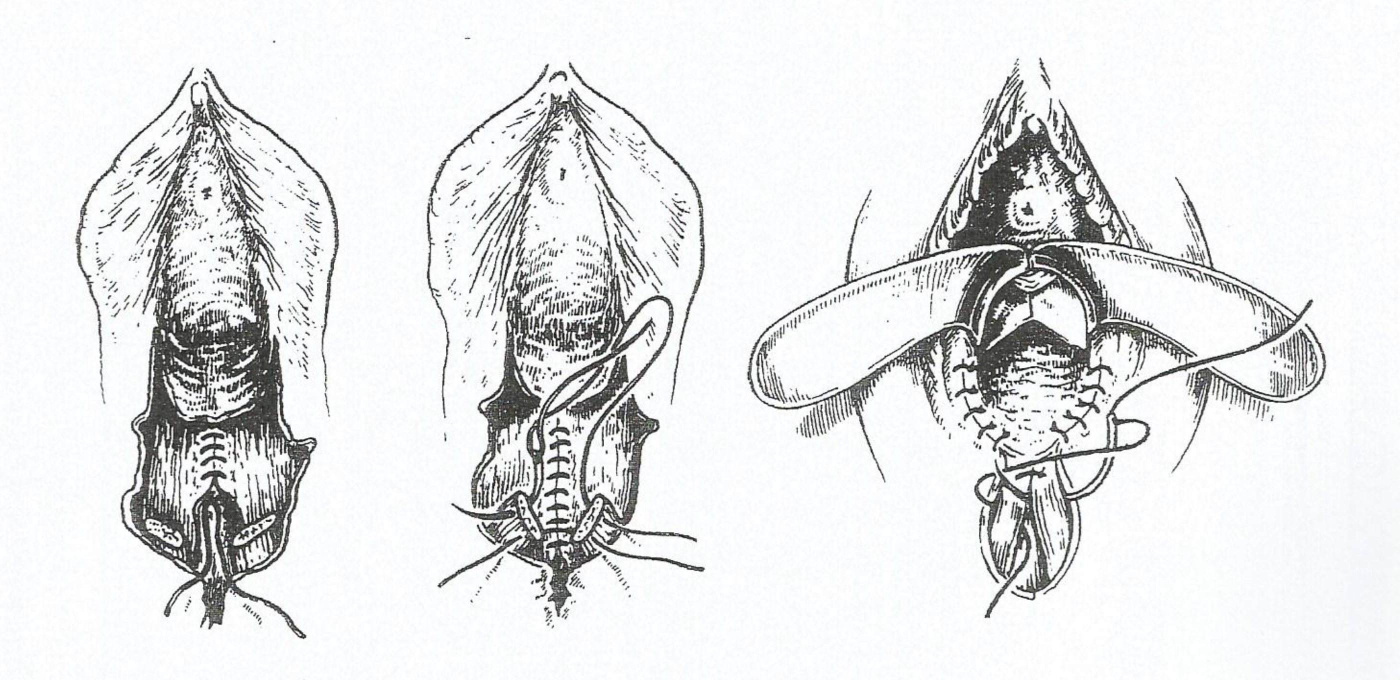
Treatment

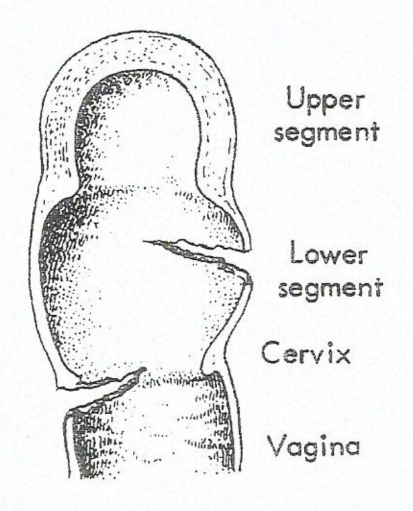
↪ **Prophylaxis** ⇨ proper management of the 2nd stage ± episiotomy (if there is overstretch or the perineum is about to tear)

↪ **Active** ⇨ immediate surgical repair (within 24–48 hrs of delivery)

↪ **Old complete perineal tear**
- If later → wait 3-6 m (tissues may be edematous ± infected)
- Anatomical repair in layers [Lawson Tait operation]


5
10
Local
infiltration
anesth.
lignocaine 1%




Upper
segment
Lower
segment
Cervix
Vagina

Technique of repair

* Local infiltration is better than GEA
* Interrupted sutures are better than continuous
* Vicryl is better than chromic catgut
* Sutures are taken from above downwards

- Rectal mucosa → INVERTED LAMBERT sutures (to avoid mucosa)
- Ext. anal sphincter → approximate the 2 dimples at sides of anus (torn ends)
- Deep perineal muscles ± Levator ani
- Vaginal wall → continuous or interrupted stitches
- Superficial perineal muscles & then → perineal skin closure

Post-operative care

- Minor degrees → local cleanliness
- Major degrees →
  - *perineum* : dry , clean , antiseptic as betadine (povidone iodine)
  - *diet* : NPO for 48 hrs, then → low residue + increased fluids
    Laxatives are used for 2 weeks (stools should be soft)
  - *systemic antibiotics*........intest. *antiseptics* (neomycin + flagyl ..5ds)
  - *no rectal* suppositories..............No *sexual* intercourse for 2-3 ms

[2] Vaginal tears

Etiology: as perineal tears

Diagnosis

- Traumatic PPhge (fresh blood +contracted uterus)
- EUA (with good light + retraction by Sim's speculum
  + Auvard self retaining post vag wall retractor)

Complications

Early → Hge (sometimes difficult to control ✓) ± infection
COLPORRHEXIS (rupture of the vaginal vault or post. fornix)

Late

- If bladder is involved → vesico-vaginal fistula or incontinence
- If rectum or sphincter → recto-vaginal fistula or incontinence
- If levator ani → prolapse
- Poor healing → vaginal stenosis → dyspareunia

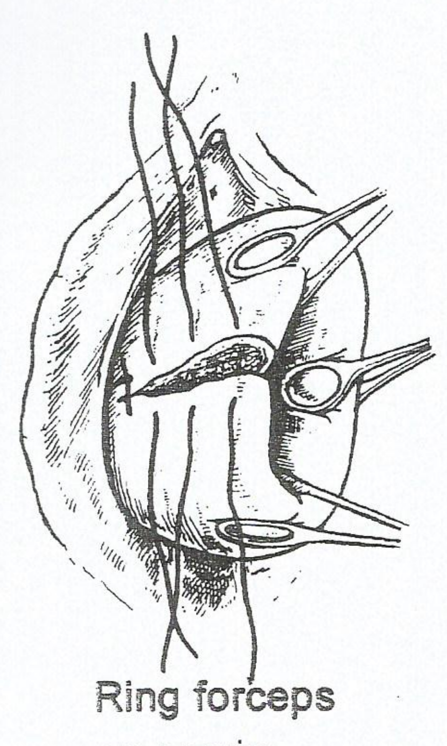
Treatment ⇨ resuscitation 1st

- Immediate repair (from above downwards)
- If failed → vaginal pack + catheter + abcs (for 24 hrs)
- If failed → bilateral internal iliac artery ligation.

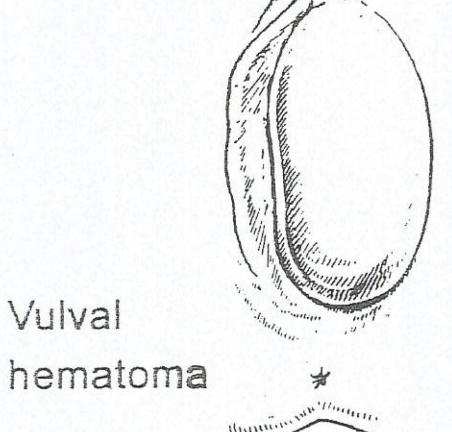
Bandl's ring

Thinned out lower segment

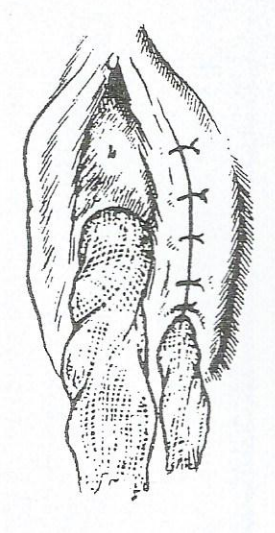
Cervix tears away from vagina here



Ring forceps on cervix



Vulval hematoma



[3] Cervical tears

Etiology:

- *causes in the passage* → cervical fibrosis
- *causes in the passenger* → large baby
- *causes in power* → ppt labor
- *obstetric operations* → forceps, ventouse , manual dilatation of cx

Types

1. *Unilateral*
2. *Bilateral*
3. *Stellate* (multiple radiating)

Diagnosed by EUA ⇨ the cx is grasped by "4" ring forceps at its 4 corners

Complications

- **Early**
  - Hge + infection
  - Rupture uterus → if it extended upwards ¤
  - Ureteric injury → during surgical repair
- **Late**
  - . Patulous internal os → habitual abortion & preterm labor
  - . Chronic cervicitis → infertility or cervical dystocia
  - . Ectropion → eversion of cervical lips in bilateral tears

Treatment ⇨ resuscitation + suturing cx tears from apex downwards
NB: she may need cerclage in next pregnancy

[4] Genital tract hematomas

Etiology ⇨ . Traumatic vaginal delivery e.g. forceps
. Sometimes occur in normal spontaneous labor ¤

☆ *Vulval (infra-levator)*

- Presence below levator ani → prevents its upward extension
- There is tense tender bluish fluctuant swelling at the vulva
- ttt ⇝ observation if small & localized

☆ *Paravaginal (supra-levator)*

- Sometimes not easily seen (felt by P/V)
- May be suspected by sense of rectal straining (due to pressure)
- ttt ⇝ evacuation only if *large* ¤ + drain + packing the vagina

☆ *Broad ligament (Sub-peritoneal hematoma)*

- Progressively expanding → broad ligamentary swelling
- It may dissect its way upwards → may even reach up to diaphragm
- ttt ⇝ laparotomy: evacuation ± bilateral uterine artery ligation

Scar rupture (bleeding is less as scar is fibrotic) ¤

USCS (2 – 4%) ¤	**LSCS** (0.2 – 0.4%)
1- Thick muscle layer → difficult to coapt edges	1- easy to coapt edges
2- Haemostasis difficult → Hematoma weakens scar	2- haemostasis more easy
3- infection more common	3- infection less common
4- Muscle is active in puerperium → pulls on scar	4- muscle less active
5- Placenta may implant on scar in next pregnancy	5- less liable

(5) Rupture uterus ✍

Incidence

- Varies according to level of obstetric care ¤ (1/1.000 → 1/4.000)
- Rupture uterus is the *worst* complication facing the obstetrician
- It should be suspected in any patient with *collapse* during or after labor
- <u>More common in MG ¤ (96%) due to</u>
    - *Passage* ⇨ weak uterine wall & pendulous abd. (→↑malpresentations)
    - *Passenger* ⇨ ↑ fetal size (&↑ % of DM)
    - *Power* ⇨ ↑↑ uterine contractions in response to obstruction ✌
    - *Attendant* ⇨ false sense of security

Etiology ΦΦ

↪ <u>During pregnancy.....APHge</u>

A- Spontaneous

- Ruptured previous uterine ✓ scar ¤ (UUS >LUS)
- Rupture of anterior sacculation → in fixed RVF
- Rupture of posterior sacculation → in ventrofixation
- Rupture of pregnancy in rudimentary horn
- Invasive trophoblastic disease
- Placenta percreta
- Concealed accidental haemorrhage

B- Traumatic

- Trauma to the abdomen (e.g. penetrating wounds, seat belts)
- External cephalic version (ECV)

↪ <u>During labor........PPHge</u>

A- Spontaneous

- Same etiology as during pregnancy (scar✓)
- Obstructed labor ✓✓ (the commonest)
- Maluse of ecbolics

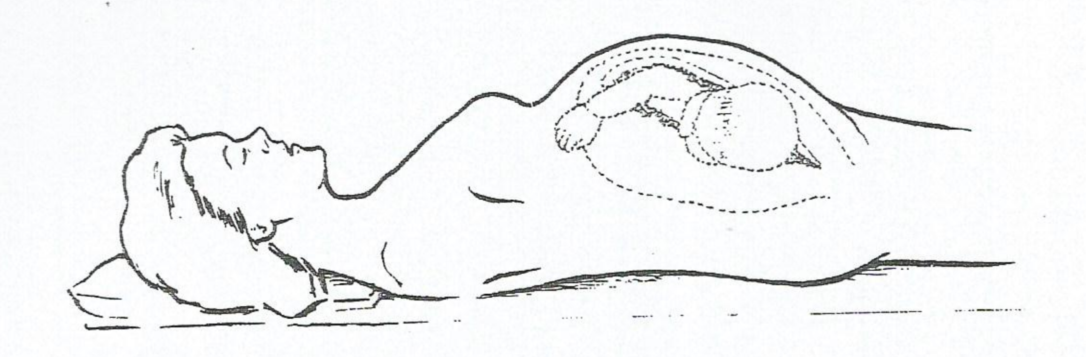
B- Traumatic

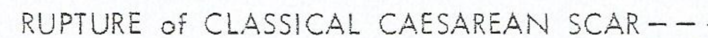
- Obstetric operations < full cx dilatation (✓forceps)
- Excessive fundal pressure
- Manual dilatation of cx or extension of a cx tear
- Manual removal of placenta ¤

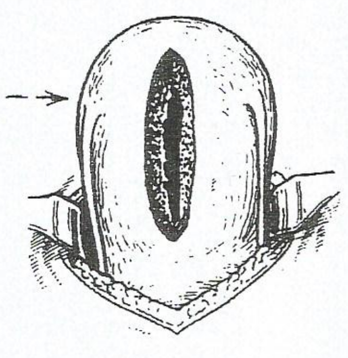
Types

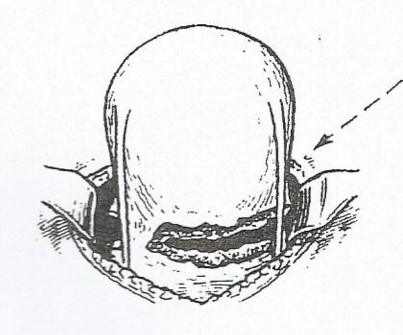
☆ <u>Complete rupture</u> (UUS) all 3 layers (including peritoneum) are ruptured
↘ massive intraperitoneal haemorrhage

☆ <u>Incomplete rupture</u> (LUS) muscle layer is only ruptured with intact peritoneum ¤
↘ subperitoneal hematoma (occult rupture) or




RUPTURE of CLASSICAL CAESAREAN SCAR




RUPTURE of a LOWER SEGMENT SCAR

SPONTANEOUS RUPTURE



Clinical Picture

✵ Rupture during pregnancy ✵

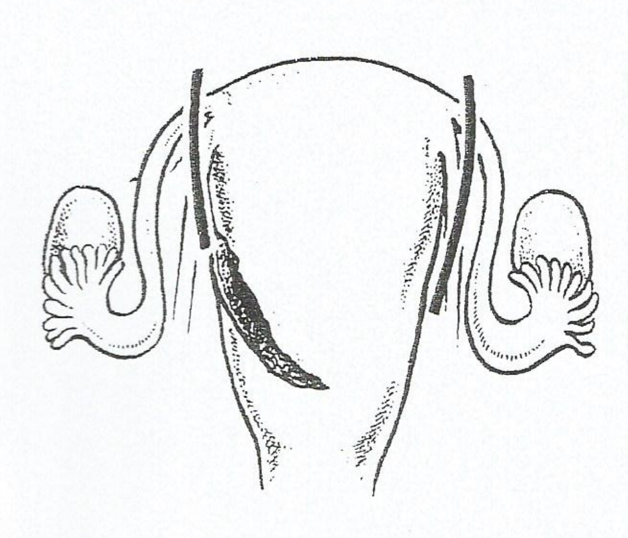
1) Impending (threatened) rupture of scar
   * Separation of the fibrosed edges of a scar
     . min. *symptoms* (tender scar) or *signs* (vag. spotting) d.t. fibrosis ¤
     . The patient may even come to hospital walking (silent rupture)
   * U/S → gapping (dehiscence) of the scar

2) Frank rupture uterus (acute abdomen)
   - *Symptoms*
     - Sudden severe abdominal pain, followed by
     - Collapse (internal hge: usually *severe* esp. if ut. vessels are torn)
   - *Signs*
     - General → Shock
     - Abdominal → . T, R , RT (Late: Cullen's sign, shifting dullness)
       . Fetus felt abdominally with –ve FHS
       . Uterus retracted away & becomes lax ¤
     - Vaginal →
       . External bleeding may be present
       . Hematuria ¤ may be present (injury of *urinary bladder)*

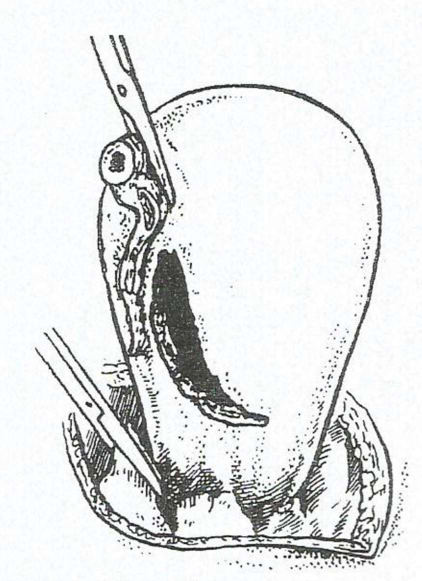
✵ Rupture during labor ✵

1) Obstructed labor
   - *Symptoms:* Of obstructed labor , then
     - Cessation of labor pain → sudden severe abdominal pain
     - Vaginal bleeding (& feeling of something giving way ¤)
     - Collapse (d.t. both vaginal & intraperitoneal hge.)
     - Cessation of fetal movements
   - *Signs*
     - General → shock + dehydration
     - Abdominal → as in preg.....
     - Vaginal → of obstructed labor . vulva : edematous
       . vagina : dry hot edematous
       . cervix : edematous or tear
     → Plus . vaginal bleeding
       . presenting part may recede upwards

2) Traumatic rupture (forceps)
   - *Suspected by* → PPhge following obstetric operation
   - *Discovered by* → Routine fundo-perineal examination



Division of the fallopian tubes and broad ligaments, leaving behind the ovaries and part of the tubes.



After incision of the peritoneum at the site of rupture the bladder is stripped from the uterine wall and a subtotal hysterectomy performed.

Differential diagnosis

1] *Bleeding according to time* (APhge , IPhge , PPhge)
2] *Acute abdomen in pregnancy or labor......................*

Complications

1] **Maternal**

* *Mortality* (10%) ➡ hypovolemic shock ± acute renal failure
* *Morbidity*
  - Infertility → if hysterectomy was done
  - Rupture uterus in next pregnancy → if repair was done
  - Ureteric injury during repair (esp. on left side)
    (as rupture is > on **Lt** side d.t. dextroratation)

2] **Fetal**

* complete rupture → 100% mortality
* incomplete rupture → 60% mortality

Treatment

▸ **Prophylaxis**

1- Proper antenatal care

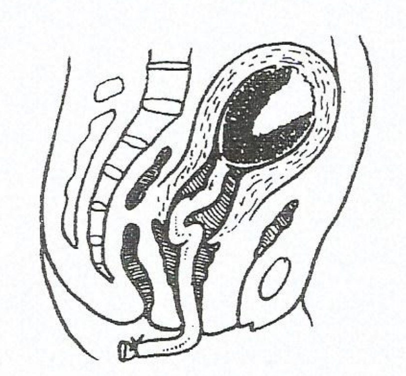
* Early detection of any abnormality needing CS (macrosomia, CPD)
* GMP must deliver in HOSPITAL (why?)
* Patient with previous uterine operations must deliver in HOSPITAL
  - One LSCS → may try vaginal delivery
  - Two or more LSCS → elective C.S. at completed 37 wks (38)
  - One USCS or hysterotomy → always C.S.
  - Previous repair of rupture should be hospitalized all-through

2- Proper intranatal care

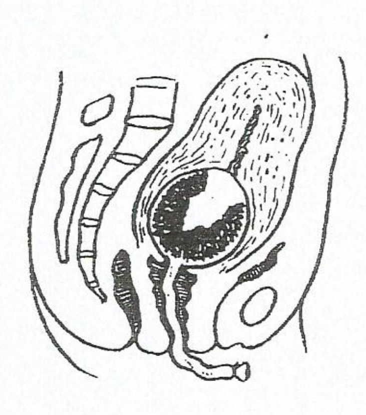
* Early detection of signs of obstructed labor
* Proper use of ecbolics
* Adequate precautions in operative obstetric deliveries
* EUA if PPhge occurred for early diagnosis

▸ **Active**

○ Resuscitation
○ Laparotomy **'midline incision'**
  - Supravaginal hysterectomy (ideal ttt) ✓✓ -LEAVE THE OVARIES-
  - Bilateral IIA ligation may be needed to control hge.
  - Exploration of injury of other structures (bladder, ureter)
○ Conservation (repair) of uterus may be done in limited cases ⇝
  - PG, young patient..........Clear cut edges, small wound
  - Patient must be hospitalized next pregnancy



RETAINED PLACENTA



③ Retained placenta

Definition (0.5 – 1%)

Failure of delivery of the placenta
within ½ hour of delivery of fetus

Physiology of placental separation

Depends on uterine contraction & retraction. The placenta being inelastic → can't cope with the ↓ in uterine length:-

❶ *Separation* of placenta at the line of cleavage between it & the uterus
❷ Followed by placental *descent* through the genital tract

Etiology ΦΦ

↪ **Retained separated**
= failure of placental descent

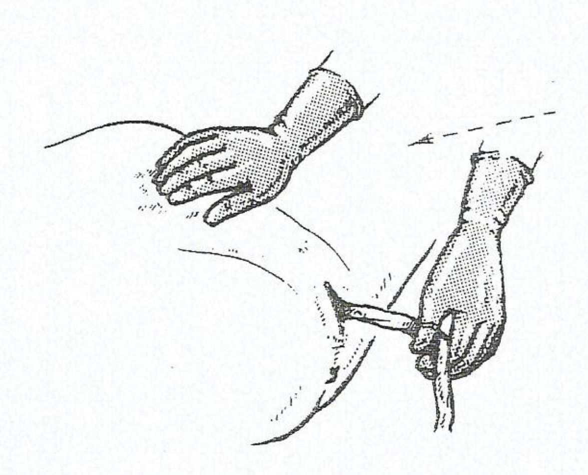
- uterine atony
- contraction (constriction) ring
- complete rupture uterus → escape of placenta to abdomen
- full urinary bladder

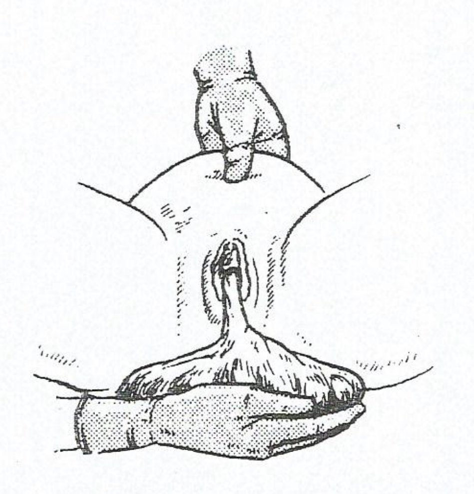
↪ **Retained adherent**
= failure of placental separation

* Simple adherence due to (uterine atony, DM, RH, syphilis)

* Morbid adherence

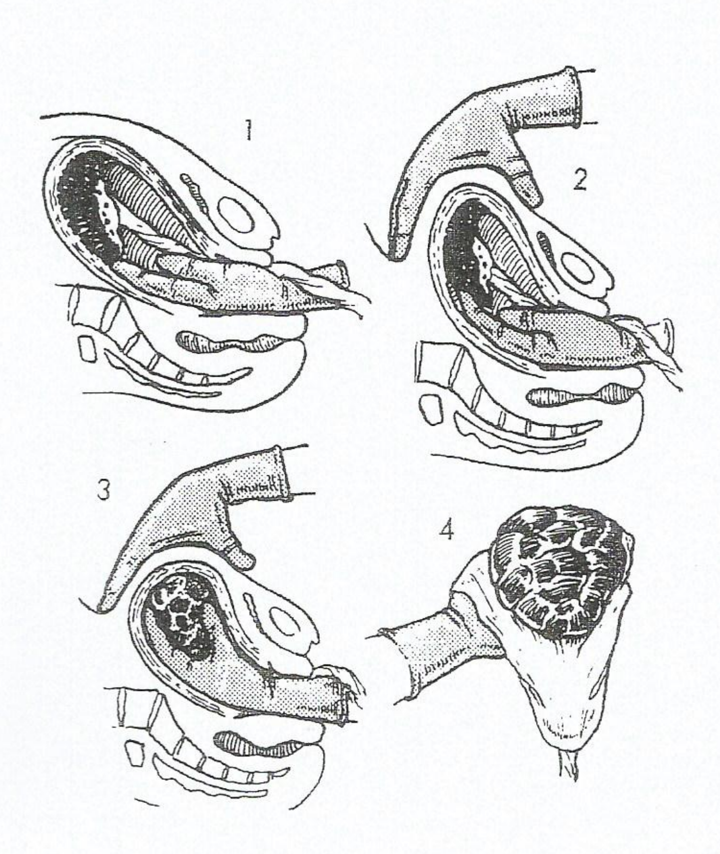
- *According to degree of invasion*
    - Placenta accreta vera: < ½ myometrial invasion
    - placenta increta: > ½ myometrial invasion
    - placenta percreta: invading peritonium even into bladder
- *According to number of cotyledons involved →*
    - adhesion may be focal , partial , total
- *Causes of adhesions (more in GMP)*
    1. Placenta previa
    2. Scar tissue due to
        - Previous C.S. , myomectomy
        - Previous manual separation of the placenta
        - Endometritis
    3. Presence of congenital uterine anomaly (as septum)
    4. Submucous fibroid
    5. Congenital absence of layer of Nitaböch





Complications of manual removal of the placenta:

1. Perforation of uterus
2. Uterine irritation → fibrous tissue
3. Retained placental fragments →
   - S_3
   - Placental polyp
   - Malignant trophoblastic disease



Clinical picture

- *History*
  - Failure of placental delivery for ½ hour
  - Bleeding →
    - If the placenta is *not* separated at all.....no bleeding
    - If it is *completely* separated..................minimal bleeding
    - If it is *partially* separated...................massive bleeding

- *Examination*
  - General → shock (hypovolemic ± neurogenic from Crede's method)
  - Abdominal →
    - Fundal level elevated above umbilicus
    - Signs of placental separation +ve or –ve
    - Uterus may be atonic

Treatment resuscitation +

▸ **Active 3rd stage management**

- Ecbolics , massage , Brandt Andrews method →
- This will deliver an unadherent placenta in the absence of contraction ring

▸ **If failed → Manual separation of placenta under anesthesia**
Introduce right hand along the cord, you may find

- *Contraction ring*
  - treat by delivering under GEA (halothane)
  - if failed → give uterine relaxant as amyl nitrite or other tocolytics
- *Rupture uterus* → laparotomy
- *Placenta adherent*
  - reach the margin (line of cleavage between placenta & uterus)
  - take a fold of membrane, separate the placenta by sawing manner
  - placenta must be fully inspected for missing parts.

▸ **If failed → morbid adherence of placenta**

- *Supravaginal hysterectomy* (ideal ttt)
- *Conservation* (if in much need of children & bleeding is not severe)
  - Cut the cord → leave the placenta or do morcellation
  - followed by → methotrexate, methergine, antibiotics
  - BUT still remains great hazard of hge & inf. →supravag.hyst.

④ DIC (consumptive coagulopathy) 

Definition

Paradoxical situation in which both ***thrombotic*** & ***fibrinolytic*** mechanisms are simultaneously activated →
both *coagulation* & *hge* are present in the same time

Pathogenesis

- Presence of certain causes (severe / persistent) → activation of coagulation
- Dissemination of thrombosis → tissue ischemia & infarction
- Consumption of platelets / clotting factors + activation of fibrinolytic system → failure of clotting system → bleeding ↻

Etiology

ΦΦ	Mechanism
1. Placental abruption ✓✓¤	○ liberation of tissue thromboplastin ○ consumption in retromembranous hematoma
2. P.I.H.	- endothelial damage → collagen exposure - placental abruption may occur
3. Amniotic Fluid embolism	○ liberation of tissue thromboplastin ○ procoagulant activity of amniotic fluid
4. Retained IUFD	- liberation of tissue thromboplastin - fibrinogen consumption in the dead fetus
5. Septic abortion (or any septic shock)	○ liberation of tissue thromboplastin ○ release of bacterial endotoxin
6. Induction of abortion	- By intramniotic injection of hypertonic saline or urea → necrosis of uterus → thromboplastin
7. Blood transfusion -excess dextran transfusion	○ forms complex with fibrinogen → inactive
-Massive transf. (dilutional)	○ esp.>5 unit or old blood → ↓coagulation factors
-incompatible transfusion	○ or massive hge & hypovolemic shock
8. Any excess tissue damage or hypoxia	- liberation of tissue thromboplastin e.g. placenta accreta , rupture uterus , hysterectomy

Clinical Picture

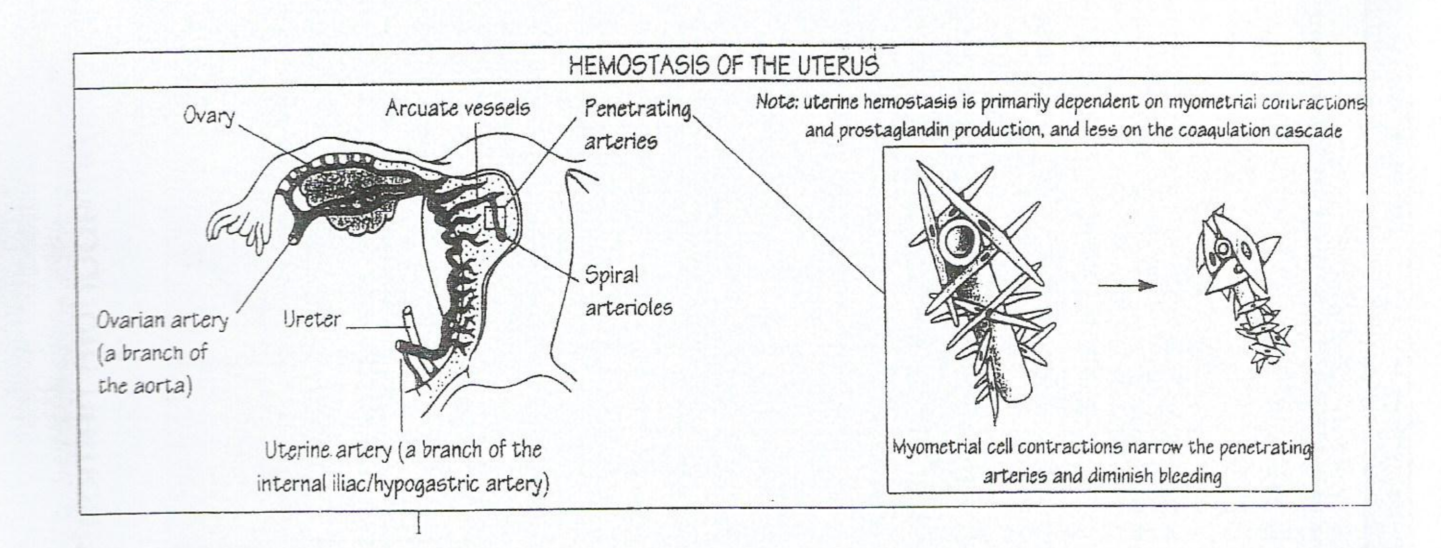
1. Proper anticipation (presence of pdf e.g. abruptio placenta)
2. Thromboembolism → pulmonary, renal, ... infarctions
3. Bleeding tendency → petechiae, *hematuria*, PPhge, ...

Coagulation factors

I	Plasma fibrinogen	VIII	Anti-haemophilic factor
II	Prothrombin	IX	Christmas factor
III	Tissue thromboplastin	X	Stuart Prower factor
IV	Calcium	XI	Pl. thromboplastin antecedent
V	Pro-accelerin (labile factor)	XII	Hageman factor
VII	Pro-convertin	XIII	Fibrin stabilizing factor

Pathways

Intrinsic pathway	**Extrinsic pathway**
Surface activation → collagen	tissue thromboplastin → activation
XII → XI → IX → X	VII → X
Assessed by PTT	assessed by PT



Investigations

↪ **Coagulation profile:**

1. Platelet count (N: 250,000/ml). thrombocytopenia is < ....
2. Fibrinogen (N: 200-300mg% - in pregnancy : 400-600mg%)
3. Fibrin degradation products (N: 10 μg/ml) in DIC > 40 μg/ml
4. D-dimers (in DIC >0.5 μg/ml)

↪ **Prolonged**

1. Bleeding time (N: 2-4 min)
2. Clotting time (N: 6-12 min)
3. Prothrombin time (N: 12 sec.)
4. Partial thromboplastin time (N: 35-45 sec)
5. Thrombin time (time needed for conversion of fibrinogen to fibrin)

↪ **Weiner test (clot observation test...poor man's clot) Bed side test**

- 5 ml blood in a test tube → clot forms in 6-12 min & dissolves >30 m
- Failure of clotting within 15 min → fibrinogen <100 mg%
- Early dissolution before 30 min → increased fibrinolysis

Treatment

[1] **Treatment of the cause**

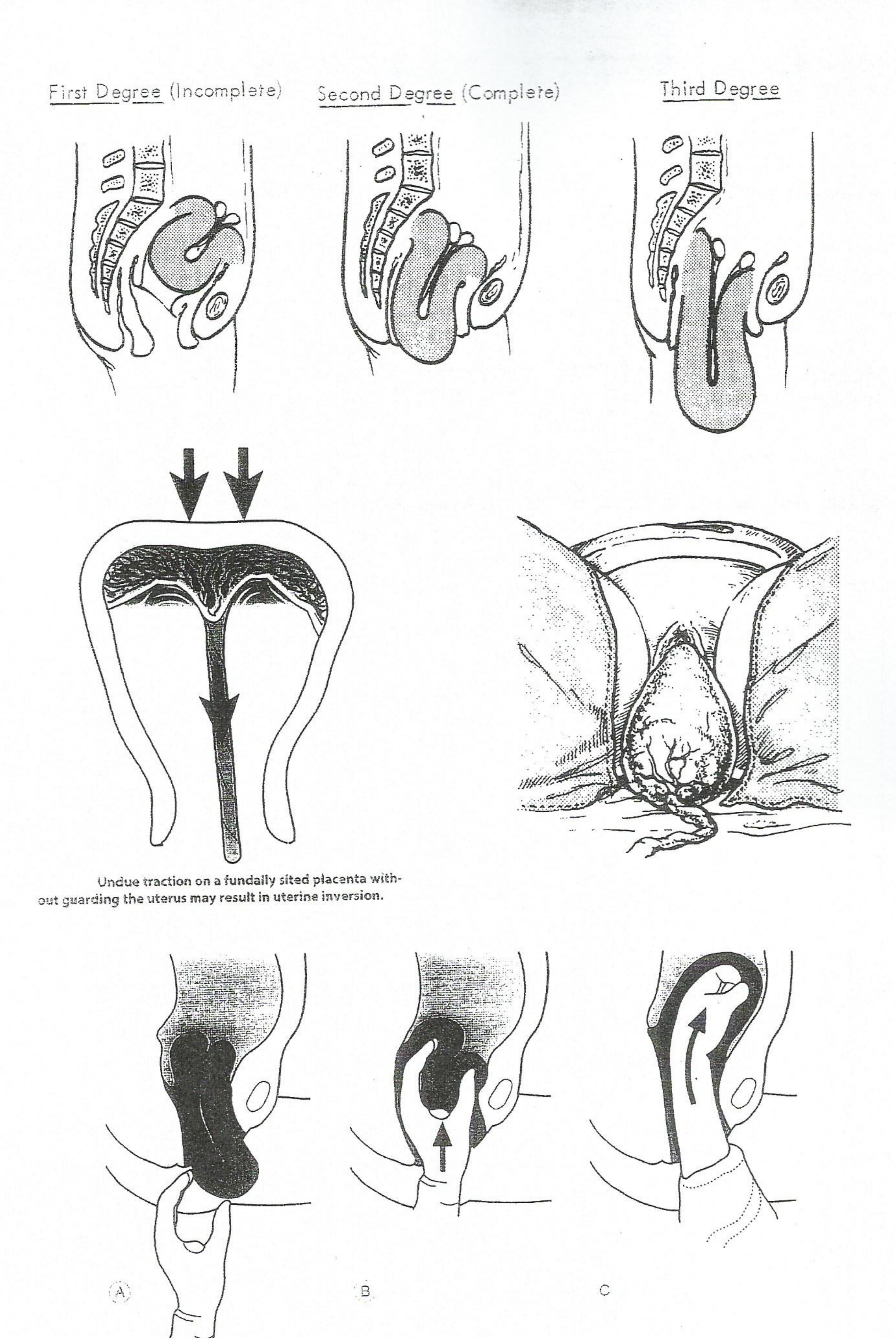
- In most cases by termination of pregnancy as early as possible
- Vaginal delivery is more *safe* although CS is more *rapid*

[2] **Life saving measures**

- Resuscitation
- Correction of coagulation defects
  - Fresh blood transfusion
  - Fresh Frozen Plasma (fibrinogen + coagulation factors)
  - Cryoprecipitate (dried fibrinogen or some coagulation factors)
  - Platelet transfusion

[3] **Don't give**

- *Heparin* → as it increases bleeding (except in IUFD: as there is intact vascular tree –the patient is not bleeding– then heparin is stopped & TOP is induced after 6 hours
- *Antifibriolytic* drugs → as it increases thrombosis (they also cross placenta to the fetus)



Undue traction on a fundally sited placenta without guarding the uterus may result in uterine inversion.

Replacing an inverted uterus. **(A)** Recognition of uterine inversion. **(B)** Replacement of the uterus through the cervix. **(C)** Restitution of the uterus.

⑤ Acute uterine inversion

Def: ⇨ condition in which the uterus is **turned inside out** immediately after labor & before cx constriction (v.rare 1/3.000 – 1/30.000)

Degrees 1st° → just cupping of the fundus
2nd° → inverted fundus protrudes through the cervix into vagina
3rd° → inverted fundus appears outside the vulva (the *complete* type)

Etiology

Iatrogenic (bad 3rd stage management) ✓✓
1. Crede's method while uterus is lax
2. Cord traction while placenta not yet separated
3. Manual removal of placenta esp. if it is adherent

Spontaneous ⇨ precipitate labor or excessive straining



Clinical Picture

↪ **History**
- * severe lower abdominal pain with continuous bearing down
- * Fullness (2nd) in or something protruding (3rd) from the vagina
- * PPhge (atony) may be minimal if
  - . Placenta is still attached
  - . In severe degrees with kinking of blood vessels

↪ **Examination**
- * General → Shock (hypovolemic & neurogenic)
- * Abdominal: . 1st degree → cup shaped fundus
  . 2nd & 3rd° → absent fundus
- * Vaginal
  - 1st → depressed fundus inside the uterus
  - 2nd → cervix surrounds inverted fundus
  - 3rd → fundus protrudes through the vulva

Differential Diagnosis
1. Causes of postpartum shock
2. Uterine prolapse (the cervix –external ostium– is found)
3. Fibroid polyp (uterine sound passes all around)

Treatment
- ▸ **Prophylaxis** ⇨ avoid pdf. + proper 3rd stage management
- ▸ **Active** ⇨ *Resuscitation + Manual reduction* (or hydrostatic ¤)
  Under GEA (halothane / amyl nitrite / tocolytics) ¤
  - First reposit the uterus then → remove the placenta ¤
  - Then → ecbolics + massage
  - Then → Pack + antibiotics

★ Amniotic fluid embolism

Incidence ➡ 1/30.0000 with 50% mortality

Etiology

- AF may enter into the maternal circulation d.t.:-
  1. <u>*Increased intrauterine pressure*</u>
     ↘ accidental hge, oxytocin overdose with intact membranes ¤
  2. <u>*Opened uterine or endocervical veins*</u>
     ↘ as in genital tract lacerations e.g. rupture uterus
- The above factors also lead to fetal distress → meconium stained AF → this potentiates the toxic nature of AF → worsens the symptoms

Pathogenesis

- *Immediately or shortly after* a difficult delivery:-
  - RDS & circulatory collapse (extensive pulmonary vascular obstruction d.t. the AF particulate matter → acute cor-pulmonale → abrupt hypoxia & CHF)
  - DIC → bleeding from genital tract & all other sites of trauma
  - Deep coma & immediate death (>50%)
- *Recently*, it is proved to be a form of <u>*anaphylactic*</u> shock to the antigenic AF (thus AF embolism is a mis.......)

Diagnosis

- *Suspected in* → any case of sudden postpartum collapse & DIC
- *Proved by* → finding AF debris (fetal squamous cells, lanugo hair, vernix) in the pulmonary vessels by <u>*autopsy*</u> ¤
- *Investigations* → ECG, chest X-ray, V-Q scan

Management

- *Very difficult* (serious > pulmonary embolism ¤) → only few cases succeed
- *Immediate transfer to ICU* ↘
  - Cardio-pulmonary support
  - Management of DIC
  - Corticosteroids
  - Monitoring different organs

★ Sudden postpartum collapse 

Obstetric causes	Non-obstetric causes
- 1ry PPhge:- rupture ✓✓,-,-,-,-	- Cardiogenic e.g. peripartum cardiomyopathy
- Eclampsia	- Cerebrovascular accidents
- Pulmonary thrombo-embolism	- Anesthetic complications e.g. Mendelson $
- Amniotic fluid embolism	- Anaphylactic shock



Resuscitation. A. Algorithm. B. Position for cardiopulmonary resuscitation.

★ Shock in obstetrics

Definition ⟹ a state of circulatory failure
ς hypotension, tissue hypo-perfusion

Etiology

- Hgic shock ⇒ bleeding in early preg., APHge, PPHge
- Hypovolemic ⇒ dehydration (hyperemesis gravidarum)
- Neurogenic ⇒ pain in early preg., pain in late preg.
- Septic ⇒ septic abortion, chorioamnionitis, puerperal sepsis
- Pul. embolism ⇒ amniotic fluid or thrombus
- Splanchnic ⇒ sudden drop of intrauterine pressure (polyhdramnios, twins)

Clinical picture

- **History suggestive of**
  - Etiology e.g..missed period + acute abdomen → disturbed ectopic
  - Pdf e.g……*preg comp* (anemia, PIH)..*labor comp* (prolonged / obstructed)
- **Examination**
  1. *General* → *shock*:- low B.Pr., subnormal temp, rapid weak pulse, pale cold clammy skin, peripheral cyanosis, oliguria
  2. *Abdominal*
     - T, R, RT → internal hge e.g. ectopic
     - Bilateral adnexal swellings → V.mole
  3. Local
     - Offensive discharge → sepsis
     - Vaginal bleeding → hgic

Treatment

- General
  - Intravenous cannula….. Analgesia (morphia 15mg IV)
  - Raise legs…… O_2 inhalation…… Warmth (but not direct, to avoid VD)
- Monitoring (by fluid input & output chart)
  - Catheterization → urine should not be < 30 ml/hr
  - CVP → kept between 8–12 cm H_2O
  - Replacement → start by available fluids till blood is ready
- Drugs
  - Vaso-pressors ± inotropics
  - Corticosteroids, correction of acidosis (Na bicarb)
  - Antibiotics (in septic shock)
- Special
  - *Disturbed ectopic*…..laparotomy & salpingectomy
  - *Acc.hge*……………TOP better vaginally
  - *Rupture uterus*…….laparotomy & supravaginal hysterectomy

★ Obstetric trauma

> Types

① Maternal

* Genital tract trauma
  - *Tissue lacerations* (perineal, vaginal, cervical, uterine)
  - *Hematoma formations* (vulval, vaginal, broad ligamentary)
  - *Tissue necrosis* (bucket handle tear of cx, necrotic fistulas)
* Non-genital tract trauma (usually d.t. forceps)
  - *Injuries of pelvic joints & bones* → rupture SP, coccyx, sacro-iliac lig.
  - *Hematoma of* → rectus abdominis muscle
    - Due to rupture of the superior deep epigastric vessels
    - More common in MP after strenuous labor efforts
    - May occur after cesarean section
    - C/P → sudden severe pain ± shock

② Fetal (esp in breech)

* *Head injury* (ICHge, fractures of the skull)
* *Peripheral nerve* (brachial plexus, facial, phrenic nerve palsy)
* *Musculo-skeletal* (fracture clavicle, other long bones)
* *Soft tissue* (sternomastoid, head, abd organs lacerations)

> Long term sequelae

Vagina	Cervix	Uterus	Levator ani
Dyspareunia	chronic infection	hysterectomy	Prolapse
Fistula	Infertility	rupture uterus in next pregnancy	Incontinence - urinary - anal
	PIO, PTL		
	cervical dystocia	ureteric comp.	

★ Comp. of 3rd stage of labor 

> *Short term*

- Causes of PPhge. including 3rd stage hge (retained placenta)
- Shock & its complications → acute renal failure & DIC
- Amniotic fluid embolism
- Complications of anesthesia & blood transfusion

> *Long term*

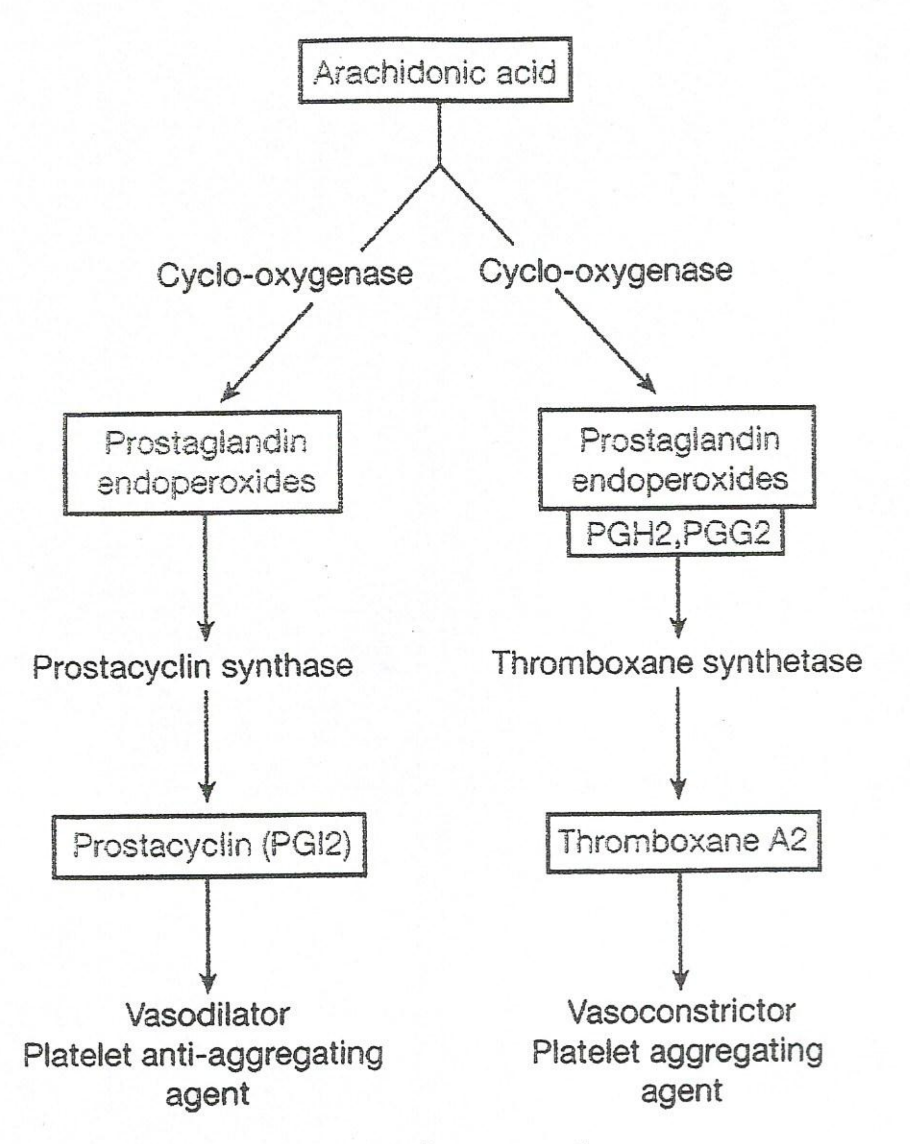
- On puerperium → $_3$S
- Infertility → due to hysterectomy or Ashermann syndrome
- Prolapse & incontinence (urinary & rectal)

Chapter 5

Abnormal Pregnancy

(Diseases With Pregnancy)

- Pre-eclampsia
- Diabetes milletus
- Heart Diseases
- Hyperemesis gravidarum
- Urinary tract infection
- Anemia
- Thromboembolism
- Thyroid disease
- Respiratory disease
- Surgery & Pain



Prostaglandin metabolism.

⚜ HTN disorders: [1] Pre-eclampsia ⚜

Definition . Occurrence of **H**ypertension, **P**roteinuria, *pathological* **E**dema
. In the **2nd** half of pregnancy in a previously **healthy** woman
. Mainly affecting PG

- **PIH** ⇔ **P**regnancy **I**nduced **H**ypertension (toxemia is a misnomer; PET ✗)
- **EPH-gestosis** ⇔ **E**dema, **P**roteinuria, **H**ypertension (by gestosis organization)

Incidence It is the commonest ✓ medical disorder in pregnancy **(5 – 10%)**

Etiology ....unknown (disease of theories)............PRIGO

[1] **Prostaglandin imbalance:** ↓ prostacyclin, $PG\text{-}E_2$ &....nitric oxides (EDRF)
↑ thromboxane ¤ & $PG\text{-}F_{2\alpha}$ &..........fibronectin

[2] **Renin-angiotensin activation** → with ↑ *sensitivity* to angiotensin II
↘ severe VC & aldosterone secretion

[3] **New theories**
- Immunological → abnormal immune response to invading trophoblast & fetal antigens (∴ it is > in PG)
- Genetic predisposition → multifactorial ? recessive ?

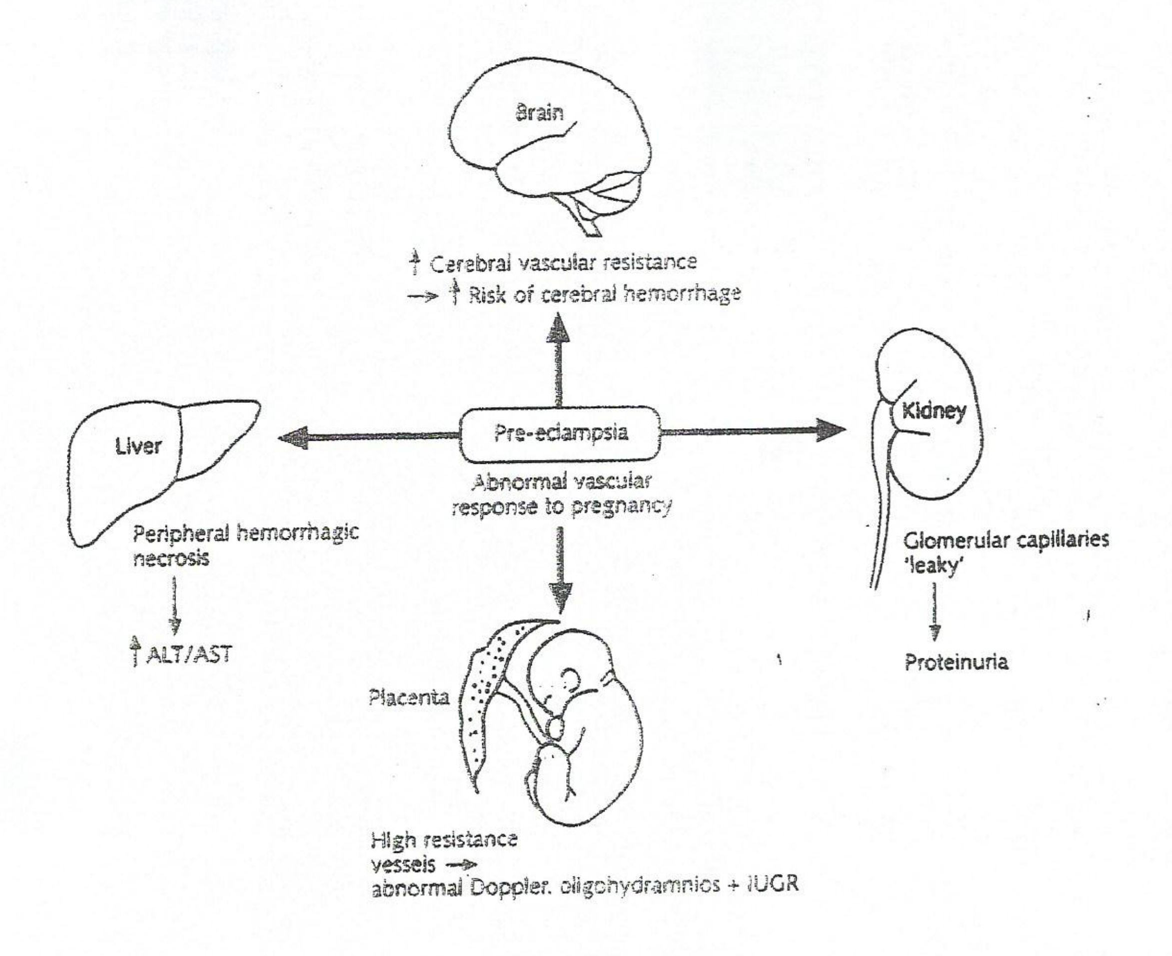
[4] **Older theories** ✗✗
- Dietary factors.............↓ vit. –↑ fat, salts (*smoking is protective!!*¤)
- Cold whether...............seasonal variation (m.b.d.t. vasoconstriction)

Predisposing factors

○ Patient ccc ⇔ (low socio-economic class)
. Extremes of age (<20 or >35)
. PG exclusively (however it may occur in MG)
. Obesity
. +ve family history

○ Obstetric disorders ⇔ PIH is more common when there is large volume of chorionic tissue
. *Twins* } It even
. *Polyhydramnios* } may occur
. *Vesicular mole* } in the 1st ½
. *Hydrops fetalis* } of preg
++ APS

○ Medical disorders ⇔ D.M. ¤, chronic hypertension, chronic nephritis, SLE



End-organ effects of pre-eclampsia.

Diagnosis of HELLP Syndrome

Hemolytic anemia

Schistocytes on peripheral blood smear

Elevated lactate dehydrogenase

Elevated total bilirubin

Elevated liver enzymes

Increase in aspartate aminotransferase

Increase in alanine aminotransferase

Low platelets

Thrombocytopenia

Main pathology

- **Vasospasm**¤ + **endothelial cell injury** → hypertension + hypoxic injury → degeneration of cells & hge
- Multiple organs are involved ∴ it is a **syndrome** (not a disease) and……HYPERTENSION….. is the *milestone* of this syndrome

Complications

………the 2nd cause of MMR…………… ✍

❶ Maternal

▸ **Immediate**

- **CNS** → eclampsia, cerebral hge / infarction, cerebral edema
- **Retina** → papilledema & retinal hge (stellate) → detachment
- **CVS** → . hypertrophy of heart (cardiomegally)
  . Up to acute HF
- **Resp** → laryngeal edema, pulmonary edema
- **Liver** → . Mainly periportal necrosis → Jaundice
  . Subcapsular (Glisson capsule) hge & rupture
- **Kidney** →
  Proliferation of *endothelial*, *epithelial*, *mesangial* cells → narrowing of the glomerular vessels → ↓ RBF & ↓ GFR
  - Damage of glomeruli → proteinuria → edema
  - Two major complications may occur
    . Renal tubular necrosis (reversible)
    . Renal cortical necrosis (irreversible)
- **Adrenal** → acute adrenal failure → Addisonian crisis
- **Metabolic** →
  - Salt & H_2O retention
  - Haemoconcentration ¤ (↓ intravascular volume ¤)
  - HELLP syndrome in severe cases
    ↘ **H**emolytic anemia, **E**levated **L**iver enz., **L**ow **P**latelet

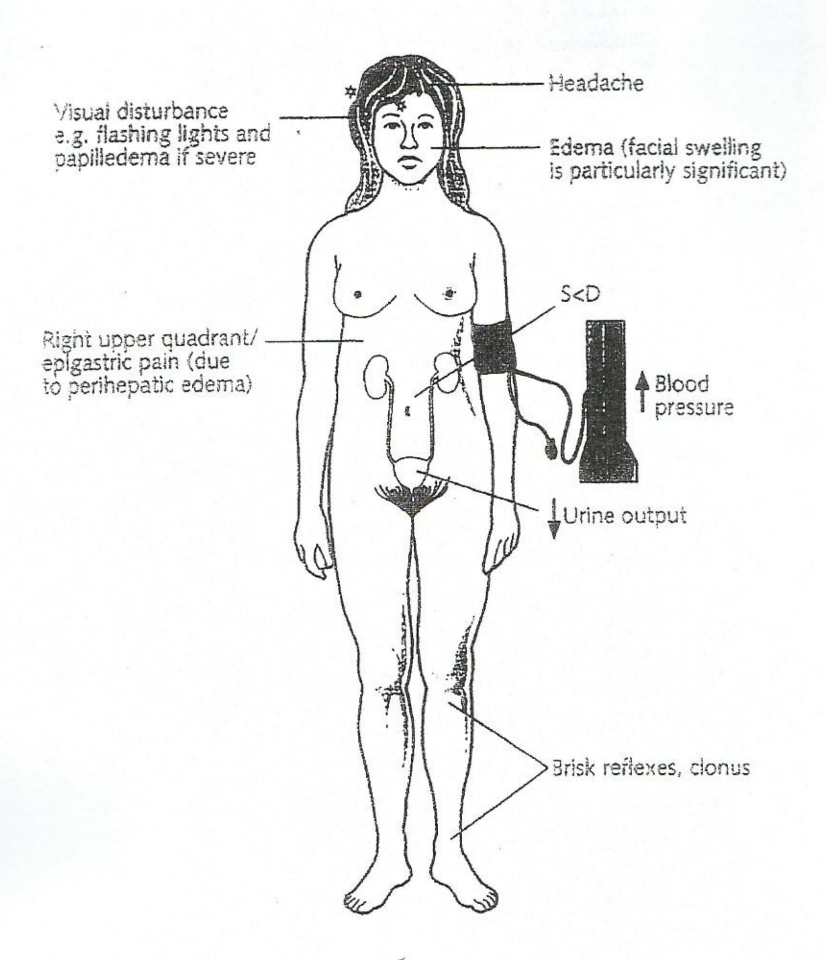
▸ **Remote**

- Residual hypertension or proteinuria (5–10%)
- Recurrence (MG) 30–50%

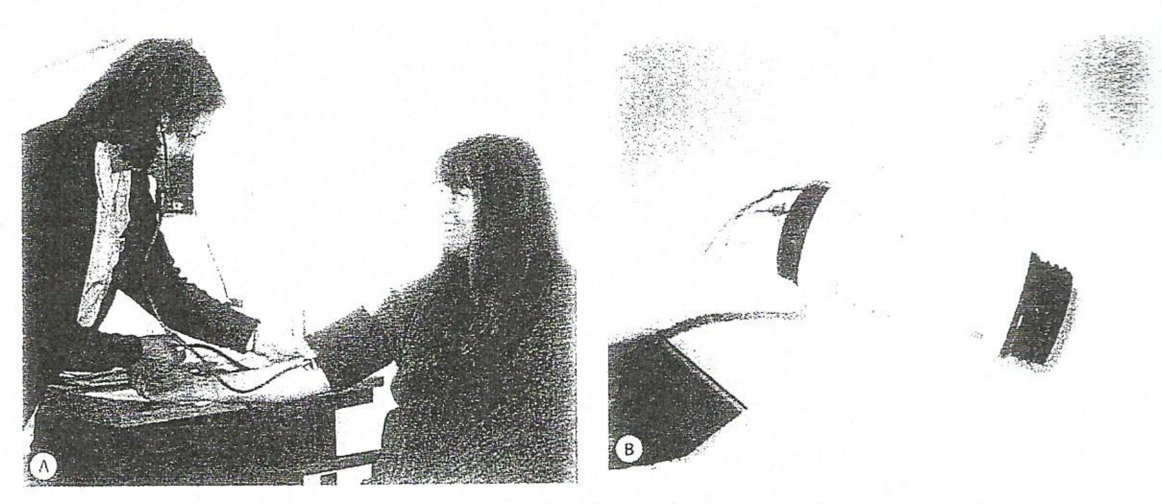
❷ Fetal & placental

▸ **Normally** → the trophoblast invades the media of the spiral vessels of decidua at 20 weeks (2ry wave of trophoblastic invasion)

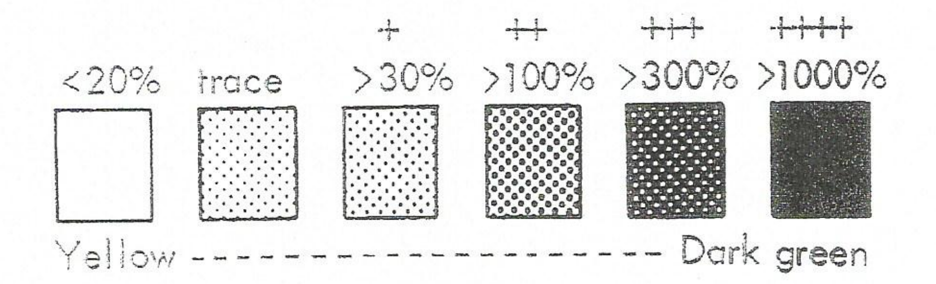
▸ **In PIH** → this is absent → the media persists → ↑ vascular resistance
↘ * IUGR & IUFD
* PTL (idiopathic or iatrogenic✓)
* Abruptio placenta → DIC



Signs and symptoms of pre-eclampsia.



Early detection of pre-eclampsia is important. (A) Measurement of blood pressure (reproduced with permission). **(B)** Testing for urinary protein.



Clinical picture

➤ **Symptoms:**.................................ONLY IN SEVERE CASES

1] NEUROLOGICAL SYMPTOMS:
- Headache (frontal, persistent, not responding to analgesics)
- Nausea & vomiting
- Visual disturbance as blurring of vision up to ↓ visual acuity

2] EPIGASTRIC PAIN → stretch of liver capsule (or subcapsular hge)

3] OLIGURIA (<400 ml /day) & ANURIA (<100 ml /day)

4] SYMPTOMS OF ANY COMPLICATION e.g. HF & Pulmonary edema

➤ **Signs:**..............................MORE IMP. & PRESENT < SYMPTOMS

1] Hypertension

- *Systolic* ≥ 140 mmHg or ↑ 30 mmHg over previous value
- *Diastolic* (✓) ≥ 90 mmHg or ↑ 15 mmHg over previous value
  ↘ Measured at semisitting ....or .... left lateral position

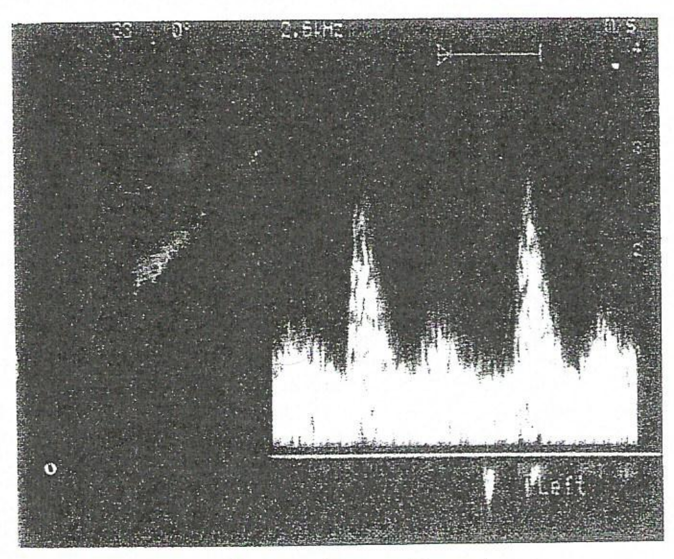
2] Proteinuria ✓

- *Non-selective*
  . A serious sign → glomerular damage
  . Detected by → albustix
- *Significant if* ≥ 300 mg/dl (1+). – normally ≈ 150 mg/dl –
  ↘ More accurate if measured in 24 hours collected urine
  ↘ Recently spot protein / creatinine ratio in a urine sample
  ↘ Proteinuria without HTN is also risky for both M & F
  ψ Mono-symptomatic gestosis (presence only of hypertension or proteinuria may occur)

3] Edema

- *Occult* ⇨ detected by rapid gain weight > 1 kg (2 pds) / 2 wks
  (Normally → < ½ kg/wk in 2nd & 3rd trimesters)
- *Manifest* ⇨ dorsum of foot, shin of tibia (m.b. normal) then become non-dependant → vulva → worsens → abdominal wall (Peau d`orange`) → swollen fingers (rings become tight) → puffy eye lids & papilledema. The worst is P.edema
- *Absent* ⇨ dry pre-eclampsia (the worst ☠)

↘ Edema is d.t. (cap. damage, hypoproteinemia, hypertension)
↘ Edema is not essential for diagnosis & has little prognostic value



Uterine artery Doppler notching at 24 weeks is predictive of pre-eclampsia and intrauterine growth restriction in high-risk mothers.

Differential diagnosis from other causes of

- **Edema**

Bilateral	Unilateral
- Physiological (at feet & ankle only)	- DVT
- Generalized anasarca e.g. H.C.R.N.A	- Varicose veins
- Endocrinal e.g. Cushing, myxedema	- Lymphedema

- **Hypertension**
  - * Pregnancy INDUCED hypertension
    - . PET (PIH)
    - . Eclampsia = (PET + convulsions)
    - . Gestational (transient) hypertension is the *appearance* of hypertension for 1st time > 20 weeks in *absence* of proteinuria & edema. It usually disappears > delivery.
  - * Pregnancy ASSOCIATED (coincidental / chronic) hypertension
    - . HTN (≥ 140/90) present < 20 weeks, or
    - . HTN 1st diagnosed in pregnancy & persists after puerperium
  - * Pregnancy AGGRAVATED hypertension
    - . Super-imposed PET (occurrence of PET on top of chronic HTN)
    - . Super-imposed eclampsia (occurrence of ecl. on top of chronic HTN)
- Proteinuria
  - * False proteinuria (the commonest cause ✓)
    - ↘ contamination from vaginal discharge; *avoided by*:
      - - Mid-stream urine sample (MSU) i.e. clean catch technique
      - - Catheter specimen ✗ (not preferable)
  - * Urinary tract infection
  - * Orthostatic (appear at end of day) → pr from lumbar spine on Lt renal vein

Investigations

1. *Renal function tests*: uric acid (1st to↑ ¤) → creatinine, urea
2. *Liver function tests*
3. *CBC* → Hct, HELLP
4. *Coagulation profile* → DIC (platelet count, antithrombin III)
5. *Fundus* → spasm, haemorrhage, exudate, edema
6. ➡ *Fetal* → FWB ✓

* Tests to detect possible development of PE [SCREENING]:
  - . **Doppler** → high vascular resistance ✓✓..early diastolic notch
  - . Roll over test (↑ BPr in supine position > 20 mmHg) 👎
  - . Cold water immersion test (↑ diastolic pr >20mmHg) 👎
  - . Angiotensin II infusion test 👎
  - . ↑ plasma fibronectin, ↓ urinary calcium 👎

Classification

- **Pre-eclampsia may be mild or severe if:-** ✍ Φ
  - *Signs* * B.Pressure [Systolic ≥ 160 mmHg – Diastolic ≥ 110 mmHg]
    * Proteinuria ≥ 500 mg/dl (++) or ≥ 5 g/L/24 hr collected urine ¤
  - *Symptoms* → appearance of any symptom esp;…. oliguria or anuria
  - *Complication* → Maternal (HELLP, DIC) or Fetal (IUGR)
  - *Investigations* → denoting any organ damage
- **Fulminating Preeclampsia: (impending eclampsia)**
  - Severe PE (symptoms, severe proteinuria) + Hyperreflexia
  - If left → may develop eclampsia [therefore ttt as eclampsia]

Treatment ✍

Prophylaxis

- **Early detection by regular ANC**✓✓ → BPr., albumin, screening tets (esp for HRG)
- **Anti-platelets** → as low dose aspirin (75mg) or juspirin (81mg)
- **May give** → vit E (anti-oxidant ¤), omega 3 (fish oil).

Mild cases

1] If mature ⇨ **Terminate**

2] Otherwise ⇨ **Conserve**

* Bed / Mental rest → sedatives in extreme cases...e.g diazepam (5 mg/day)
* Diet → balanced i.e. → avoid excess [salt, fats, CHO], not salt restriction
* Antihypertensives (*some say no need: mild case*)

	Action	Dose
α-Methyldopa (Aldomet) The most safe & widely used in mild cases ✓	Central action ¤ (acts as a false transmitter in the brain → ↓ noradrenaline).	250 mg 1x4 --up to-- **2**–4 gm /day
β-blockers (alone or + ↑)	used with caution → it ↓ placental flow & FWB (used with caution)	10 mg --up to-- 40 mg /day
Nefidipine (Adalat)		

* Observation
  - *Daily* → FHS / 6 hrs…….BPr…………………..Albuminuria
  - *Weekly* → FWB………….RFT, LFT, fundus….weight (for edema)

> ➤ Corticosteroids may be given to enhance lung maturity
> ➤ Conservation is continued till maturity (37–8 wks) unless
>   - Disease → *severe* PET
>   - Mother → *distress* e.g. HELLP syndrome
>   - Fetus → *distress* e.g. abnormal CTG, IUGR

✤ Severe cases...TOP ✤

....The only cure ✓ in spite of fetal maturity...

[1] Hospitalization

➠ **Eclampsia room or Obstetric-ICU**

Patient lies on her side in a semi-dark quiet room with available → O_2 supply, mouth gag, tongue depressor, endotracheal tube, suction machine, *anticonvulsant drugs started immediately*

➠ **Observation for**

- Vital signs → BP, P, T, respiratory rate
- Level of consciousness
- Fluid intake & urine output → chart

➠ **During fit** (emergency ttt...even done at home) → insert mouth gag, avoid biting tongue, place on her side to prevent aspiration

[2] Anticonvulsant therapy (to control & prevent further fits)

Magnesium sulfate [$MgSO_4.7H_2O$]

* **Route**

- **IV:** ✓✓ 4–6 gm slowly (over 15-20 m) then............1-2 gm/hr by drip
- **IM:** loading 14 gm (4 IV + 10 IM –5 gm/ buttock–).....then 5 gm / 4 hrs
  ↘ better avoided → sterile abscess / very painful

* **Action**

- Peripheral skeletal muscle relaxant (↓ A.Ch & Ca^{++} at NMJ) ✓
- *MILD* Subcortical depressant
- *MILD Transient* hypotensive effect [vasodilator + diuretic]

* **Toxicity Signs**

- Absent knee reflexes...............................8–12 mEq/L
- Respiratory depression............................12–15 mEq/L
- Cardiac depression................................30 mEq/L
- On high level.........................neonatal resp. depression



Due to this narrow safety margin (4–7 mEq/L), the following must be checked before each dose ➲

~ Knee jerk (patellar reflex) is still present
~ Respiratory rate not < 16 /min
~ Urine > 30 ml / hr (the only way for excretion)
~ Or the best → measure serum Mg level

* **Duration** ⇨ continue therapy for 24-48 hrs after *delivery* or the last *fit*

* **Antidote** ⇨ Ca^{++} gluconate slowly 10ml 10% solution ± O_2 ± intubation

* **In resistant cases** ⇨ Phenytoin....or....Pentothal Na (Intraval)

[3] Antihypertensive therapy

- *The aim is* to prevent maternal intracranial hge or HF; but keep diastolic BPr between 90-100 mmHg (to avoid ↓ placental bl. Flow → IUFD) ↓

	Action	Dose
Hydralazine (Apresoline) Drug of choice in Severe cases ¤	Direct arteriolar VD ↑ COP, ↑ renal flow	10 mg IV bolus repeated at 15 min intervals
Labetalol (Trandate)	α and β blocker It crosses placenta → fetal bradycardia. Also contraindicated in pts with 1st° HT block	
Nefidipine (Adalat) Sublingual	Potent drugs with rapid action → sudden severe hypotension → life threatening for mother (cerebral hypo-perfusion) & fetus (↓ utero-placental flow). Diazoxide also causes neonatal hyperglycemia	
Diazoxide (IV) most dangerous		
……*Others*……..	○ Nipride (Na <u>ni</u>tro<u>pr</u>uss<u>ide</u>) ✔ ○ Tridil (Ni<u>tr</u>oglycer<u>i</u>ne) ✔	

- *Plasma volume expansion* ⇨ given with extreme caution (volume overload)
- *NO Diuretics* ⇨ ↑ hemoconc. & electrolyte imbalance (except in HF or P.edema)

[4] Termination ✓✓✓

- Induction / augmentation of labor: 🔑+ if delivery is expected soon
  - * By AROM & syntocinon
    - <u>1st stage</u> → *intrapartum continuous fetal monitoring*
    - <u>2nd stage</u> → *shorten 2nd stage by forceps*
    - <u>3rd stage</u> → *avoid ergometrine after delivery (oxytocin may be given)*
- Cesarean section: 🔑 but first correct the general condition (anti-HTN, $MgSO_4$, correction of the *severe* metabolic acidosis due to fits)

[5] Treatment of complications

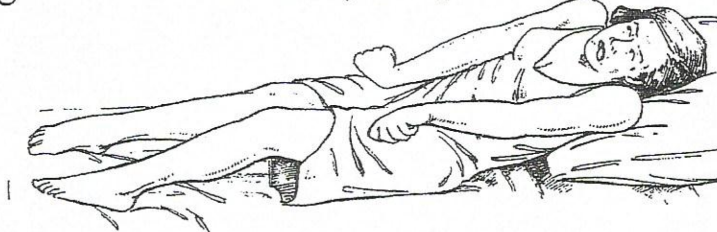
- Maternal…….renal shut down, HELLP
- Fetal………..IUGR

[6] Postpartum

- Anticonvulsant therapy continued for 24–48 hrs after….
- Antihypertensive therapy may be given if needed
- Screening for PIH in next pregnancy (became high risk)

Eclampsia

Definition ⇒ occurrence of fits (grand-mal-seizures ¤) in a patient with PET



Etiology

- Cerebral irritation by edema **or** electrolyte imbalance (↑Na^+)
- Cerebral ischemic foci by vasospasm **or** platelet thrombi

Clinical picture ....C/P of impending eclampsia → Fits...............

- Stages of fits
  1] Premonitory / Prodroma (3–5 min)
  . Twitches in muscles of eyes or face, rolling of eyes
  . Severe headache, disturbed consciousness
  2] Tonic phase (30 sec)
  All muscles of body pass into spasm. Back is arched (episthotonos), limbs stretched, respiration stops → cyanosis
  3] Clonic phase
  Intermittent contraction & relaxation of muscles → biting of tongue, vomiting, aspiration, spontaneous defecation or micturition, stertorous breathing, falling from bed → fractures
  4] Coma stage (d.t. severe acidosis)
  Variable → may recover <u>OR</u> pass into another fit (recurrent or status eclampticus) <u>OR</u> dies without recovery

- Types of fits
  Antepartum eclampsia → 70% ¤
  Intrapartum eclampsia → 20%
  Postpartum eclampsia → 10% (worst ☠), during 1st 48 hours up to....
  (the disease process is continuing though pregnancy has ended)

Differential diagnosis

Convulsions

- *Cerebral*: epilepsy (similar!!), ICHhge, infection, tumor, trauma
- *Metabolic*: hypo- or hyperglycemia, hypocalcemia (tetany) / Tetanus
- *Poisoning* by strychnine
- Hysterical ✓

Coma

- *Cerebral......metabolic.........poisoning*
- Organ failure as uremia or hepatic failure

Complications ΦΦΦ

❶ **Maternal** (MMR ⇨ 10%)

- Complication of convulsions
  - ☆ Asphyxia due to
    - Tonic contraction of respiratory muscles
    - Inhalation of vomitus
    - Inhalation of blood from bitten tongue → aspiration pneumonia
    - Tongue falls backwards
  - ☆ Severe metabolic acidosis
  - ☆ Hyperpyrexia
- Complications of PET
  - ☆ Organ failure e.g. heart, renal, suprarenal, hepatic failure
  - ☆ Haemorrhage in vital organs e.g. IChge, abruptio placenta

❷ **Fetal** (high PNMR ⇨ 30%)

- IUGR (placental insufficiency)
- PTL (spontaneous or iatrogenic)
- IUFD (accidental hemorrhage, maternal hypoxia in fits)

♣ Bad prognostic signs (Eden's criteria)

1] **Fits** → recurrent (esp >6) & postpartum

2] **Coma** → long (esp > 6 hrs) & deep

3] **Vital data** * BPr: Systolic ≥ 160 mmHg – Diastolic ≥ 110 mmHg
* Pulse >120 ……Temp > 38°c………RR > 40 /min

4] **Oliguria, Anuria**

5] **Dry eclampsia**

6] **Organ damage** → HELLP syndrome (olive green jaundice).. S&S

Management ………..the same lines as in severe PET………..

- Eclampsia room….
- Drugs
  - Anti-hypertensives
  - Mg-SO4
- Examination ⇨ TOP
  - If favorable (Bishop > 8) → induce labor
  - If not favorable (Bishop < 8) → CS (after correction of acidosis)
- Care of complications
  - Maternal
  - Fetal

Summary of the management of pregnancy-induced hypertension

Classification	Nursing responsibilities	Obstetricians' treatment
Potential PIH	Report significant rise in blood pressure, or excessive weight gain, to obstetrician.	Usually no treatment required. See patient in 7 days.
Mild PIH	Report rise in blood pressure or excessive weight gain to obstetrician.	Possible admission to hospital, depending on socio-economic conditions. If not admitted, see patient in 3 days.
Moderate PIH	In hospital: Four-hourly recording of the blood pressure. Twice-daily urine testing for protein. Regular observation of the patient's condition, including fluid intake and output. Bed rest, but toilet privileges allowed.	Admit to hospital. Sedation (if indicated). Labetalol (starting) 100mg twice daily or atenolol (starting) 100mg in evening or oxprenolol (starting) 20mg three times daily or methyidopa (starting) 250mg three times daily. Nifedipine 10mg sublingually repeated as needed.
Severe PIH	Two-hourly blood pressure recording for 6 hours, then 4-hourly. Urine testing for protein and acetone twice daily. Fluid intake and output recorded. Careful observation of the patient for the signs of imminent eclampsia. Complete bed rest for 24 hours, thereafter possible toilet privileges.	Admit to hospital. Depending on the severity of the illness give: a) Magnesium sulphate (see page 126). This is the preferred medication b) Hydralazine intravenously Intravenous frusemide 20 mg or stable plasma protein substitute (SPPS), if persistent oliguria. ?Caesarean section.
Imminent eclampsia	The patient requires careful systematic observation as eclampsia is a possible outcome. The blood pressure requires frequent estimation, at intervals determined by the obstetrician. Fluid intake and urinary output must be measured meticulously, and the urine tested quantitatively for protein.	Magnesium sulphate (see page 126). This is the preferred medication. Hydralazine intravenously. Caesarean section.

[2] Chronic hypertension

Definition ⇨ presence of HTN < pregnancy….or….< 20 wks

- *Primary* (essential) ✓✓
- *Secondary* e.g. Renal, Pheochromocytoma, Cushing syndrome, Conn's disease, Coarctation of aorta, Thyrotoxicosis

Clinical picture

* PDF

- Old age, high parity, +ve family history
- Cardiovascular risk factors: smoking, ↑plasma lipids, obesity, DM

* SYMPTOM-LESS. Ask for ?

- Duration of hypertension before pregnancy & ttt received
- Previous complications
  . *Medical*: stroke, myocardial infarction
  . *Pregnancy*: superimposed PET, IUGR, IUFD

* SIGNS

- Hypertension:
  1) < 20 weeks, or
  2) if it persists after puerperium

Grade	Diastolic BPr.
Mild	90 – 105
Moderate	105 – 115
Severe	≥ 115

- No edema or proteinuria (unless complicated by superimposed PE or HF)

Investigations ....reflect chronicity..........

- ECG changes & cardiomegally
- Renal functions → creatinine clearance
- Fundus → atherosclerosis + hge

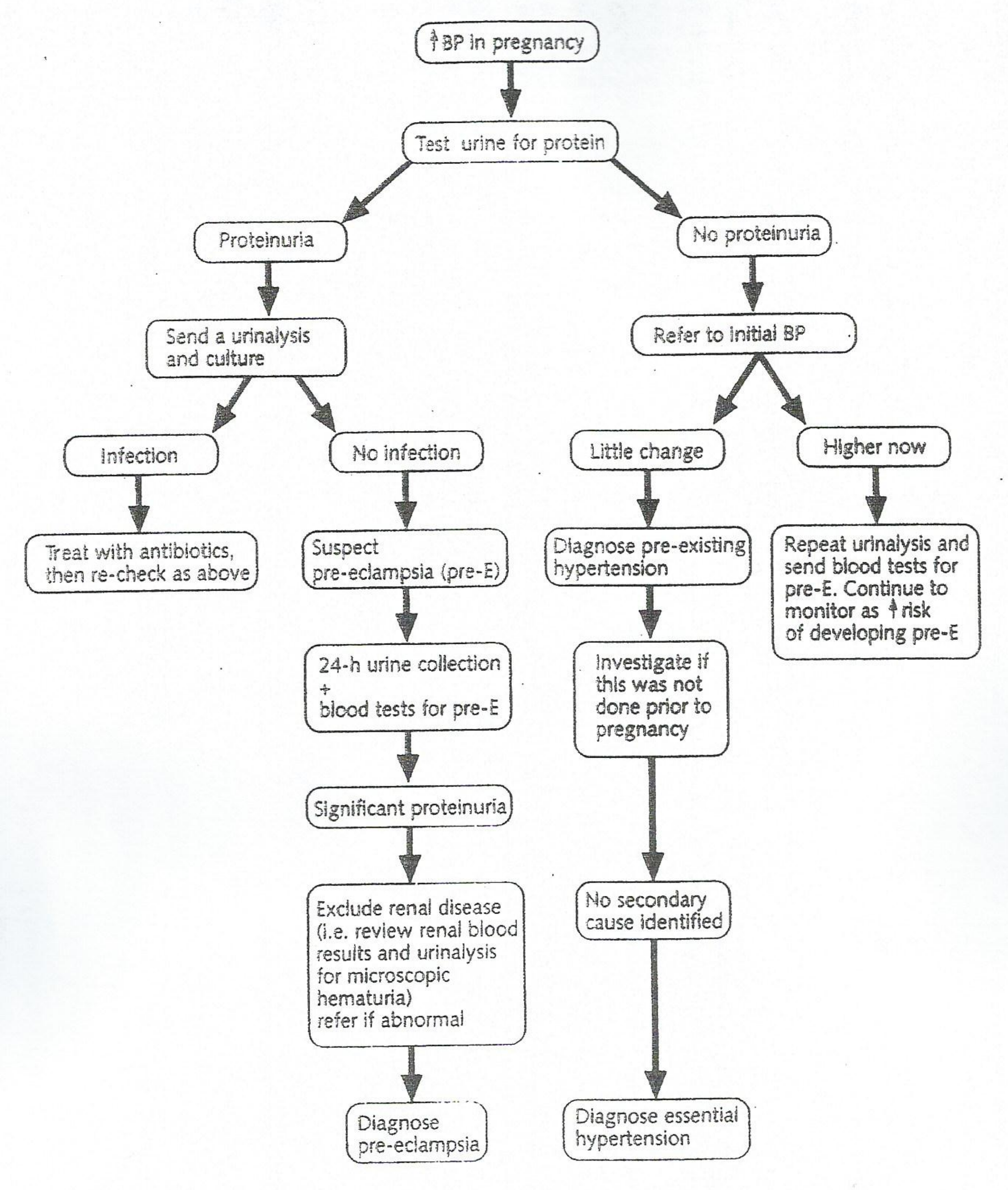
Complications

[1] Effect of pregnancy ⇨ hypertension

* Increased severity
* Superimposed PE when ⇔
- Blood Pressure (S: > 30 mmHg / D: > 15 mmHg)
- Proteinuria ± Edema
- Appearance of complications *peculiar* ✓✓ to PET (M. or F.)
- Lower 24 hr urinary Ca^{++} (40 mg) than in chronic HTN (220 mg)

[2] Effect of hypertension ⇨ pregnancy

* Maternal → all complications of PE esp Accidental hge.
* Fetal → IUGR, IUFD, PTL



Algorithm for hypertension in pregnancy.

Treatment

- **Mild** ⇔ *conservation*
  - ☆ Antihypertensives
    - 1st choice → **Methyldopa**: check liver enzymes /trim. (liver affection)
    - 2nd choice → **Hydralazine**
      - .Tachyphylaxis ¤
      - . If > 200 mg/d for > 6m → lupus like syndrome ¤, fluid retention
    - 3rd choice → **Labetalol** (200 mg tab 1x3 up to max 1.6-2.4 gm/d)
  - ☆ Used with caution
    - **Nifedipine**... safe, but may → acute hypotension
    - **Clonidine**.... safe, but acute withdrawal → hypertension
  - ☆ Contraindicated
    - **ACE** inhibitors..... fetal renal failure ¤
    - **Diuretics**... except in severe cases (heart failure)
- **Severe**
  - Medical treatment → good response → continue
  - Failed medical control → terminate

	PET	Chronic HTN	Chronic nephritis
Incidence	75%	20%	5%
Past history	– ve	< pregnancy	< pregnancy
Hypertension	> 20 wks	< 20 wks	< 20 wks
Edema	+ ve	– ve	+ ve
Proteinuria	+ ve	– ve	+ ve
ECG changes	– ve	+ ve	– ve
Renal function	⇩ if severe	*affected* with time	impaired
Fundus	⇩ if severe	*sclerotic* with time	albuminuric nephritis
TTT	TOP if severe	...according to degree of M & F affection...	
Sequlae	Recur in 30%	..condition persists & usually deteriorates...	

DM with Pregnancy 2-3%

Definition Chronic metabolic disorder of CHO metabolism
Due to *absolute* or *relative* decrease in insulin
in response to CHO challenge → hyperglycemia

★ Biphasic Glucose Control In Pregnancy

- First half of Pregnancy
  - ⇒ improved glucose tolerance due to:
    - ↑ed insulin response to a glucose load
    - ↓ gluconeogenesis & ↑ glycogen deposition
- Second half of Pregnancy
  - ⇒ increased insulin resistance, therefore:
    - 90% of DM with preg are GDM
    - GDM is not clinically apparent until ....wks
      - ∴ Screening is better done at this time
      - ∴ Patients with GDM escape CFMF

Classifications of D.M

1) According to onset

	Type I (10%) (Juvenile onset, IDDM)	Type II (90%) (maturity, non IDDM)
Etiology	*Autoimmune* (island cells anti--bodies e.g. viral inf.)	*Familial tendency* (complex & multi--factorial etiology)
Insulin	↓ (defect in pancreas)	↑ (insulin resistance)
Weight	Thin	Obese
Comp.	More e.g. DKA	Less

2) According to stages

> Potential Diabetes *patient is not diabetic but there is ↑ed risk esp. if:-*
- Positive family history (parents or her twin)
- Previous delivery of macrosomic or malformed fetus
- Parity (GMP ≥ 5) or obesity (>120% of ideal body weight)

> Latent Diabetes
The patient is not diabetic but on exposure to *stress* or *corticosteroids* ⇒ GTT is diabetic (∴ GDM is one of its forms)

> Chemical Diabetes ⇒ asymptomatic patient but GTT is diabetic (IGT)

> Established diabetes ⇒ clinical diabetes (all < that is the Pre-Diabetic state)

Complications of D.M

Effects of pregnancy ⇨ D.M.

❶ **Potentially diabetogenic + worsens established D.M.** due to
- Anti-insulin hormones (HPL, E, Pr., corticosteroids , prolactin)
- Insulinase activity in placenta

❷ **Preexisting complications may become aggravated** with increased liability for DKA

❸ **Difficult control with liability to hypoglycemia during:**
- PREGNANCY
  - Renal glucosuria (↑RBF→ ↑GFR→ ↓renal threshold to 150 mg%)
  - Alimentary glucosuria
  - Morning sickness & vomiting → starvation ketosis
  - Glucose passes to the fetus by *facilitated diffusion*... ¤
- LABOR (d.t. uterine activity)
- PUERPERIUM
  - Loss of placental hormones
  - Breast uses glucose to produce *lactose*

Effect of D.M. ⇨ pregnancy

❶ **Maternal**

- PREGNANCY
  - Preeclampsia......................in 25% ¤ (vasculopathy)
  - Polyhydramnios.................in 25% of cases (large placenta.....fetal polyuria....Anencephaly)
  - Preterm labor.....overdistension d.t. macrosomia & polyhdramnios
  - Placenta previa & abruptio placenta (PIH)
  - Pyelonephritis (recurrent) .....↑ liability to infections as candidiasis
- PARTURITION
  - PROM → fetal & maternal infection
  - Prolonged labor (d.t. macrosomia → obstructed labor → rupture ut.)
- PUERPERIUM ....S^3
  - Postpartum hemorrhage (atonic, traumatic)
  - Puerperal sepsis
  - Pulmonary embolism (obesity, vasculopathy, difficult labor)

Congenital anomalies in infants of diabetic mothers

Cardiac
- atrial septal defect
- ventricular septal defect
- coarctation of aorta
- transposition of great vessels

Other
- single umbilical artery

Gastrointestinal
- anorectal atresia
- duodenal atresia
- tracheo-esophageal fistula

Skeletal and central nervous system
- anencephaly
- caudal regression syndrome (very rare, but highly specific for diabetes mellitus)
- microcephaly
- neural tube defects

Renal
- hydronephrosis
- renal agenesis
- ureteral duplication
- polycystic kidneys

❷ Fetal

- Abortion.....↑ 3x (if uncontrolled DM) – how?
- CFMF........↑ 3x (6–9% versus 2–3% in N)
  - Especially if HbA_{1c} is increased
  - The commonest are :
    - . CVS ✓ (10x) → **VSD,** transposition of great vessels, coarctation of aorta
    - . CNS (5x) → **anencephaly**, spina bifida, meningocele
    - . GIT.....renal.....skeletal
  - A rare but very specific (pathognomonic) malformation is *caudal regression syndrome* (*sacral agenesis* ¤). This is disproved now.
- IUGR in 20% of cases (*placental insufficiency* d.t. the *vascular* changes)
- Fetal macrosomia (40% of cases)
  - Due to increased glucose in mother → hyperglycemia in the fetus → ↑ insulin from fetus → islet cell hyperplasia → marked anabolic effect. There is associated stimulation of adrenal cortex → ↑ed steroids → Na & H_2O retention
  - Newborn is *large heavy plethoric fatty* with *cushingoid* features
  - All this makes the baby more liable to all listed complications......
- IUFD due to
  - Hyperglycemia ± ketosis or Hypoglycemia
  - Vascular affection → chronic placental insufficiency & PIH
  - Congenital malformation
  - Unexplained sudden IUFD (usually after 36 weeks, repeats at same time)

❸ Neonatal ΦΦ

3 ↓ - RDS (insulin antagonizes action of corticosteroids on lung → ↓ surfactant)
↘ esp. Phosphatidyl glycerol
- Hypoglycemia (due to the ↑ed fetal insulin production)
- Hypocalcemia & hypomagnesemia → tetany

3 ↑ . Polycythemia → d.t. chronic hypoxia → erythropoietin
. Hyperbilirubinemia d.t. → prematurity, polycythemia, *oxytocin*
. Hyperviscosity syndrome → renal vein thrombosis

3 ⇒ Birth trauma → shoulder dystocia (wider than the head) + infection

CFMF → the most common cause (40%) of PNMR ✓ ¤

PNMR (4–10 %) d.t. all the above ↓ causes

nvestigations

- DM is diagnosed for the 1st time in pregnancy in 90% of cases
- History (present, past, family, obstetric) may be suggestive but investigations are a must……(as symptoms are query: pppp)

❶ Screening

1) Glucose in urine (the worst) X
   * Done by: Benedict / Fehling test….or glucose strips (easier)
   * Inaccurate as glucosuria could be due to
     - Renal glucosuria
     - Alimentary glucosuria
     - Lactosuria (some breast lactose → escape in urine)

2) Fasting blood glucose (N: <105 mg/dl)….. ideal 60-90
3) One hour postprandial (N: <140 mg/dl) the best
4) Two hours postprandial (N: < 120 mg/dl)
5) Random blood sugar (N: < 200 mg/dl)

➲ The best screening test is 1hr-PPS ✓✓
(Glucola test –50gm–)

- Time
  - For all patients (low-risk) → at 24–28 wks (universal screening i.e. done for all pregnant women without C/O)
  - For high-risk groups → at booking (1st antenatal visit):
    1. Maternal obesity or age > 35 yrs
    2. Chronic hypertension / renal disease
    3. Positive family history
    4. History of
       , GDM / IGT
       , Fetal macrosomia
       , Idiopathic polyhdramnios
       , CFMF
       , Unexplained IUFD

- Result
  < 140 mg % → no further investigations or ttt
  > 140 mg % → 3 hr GTT

Modified Priscilla White classification

	Onset	Duration	Complications	
A	**A**symptomatic but with diabetic GTT .....A_1 : FBS <105 (→ diet) .....A_2 : FBS > 105 (→ insulin)			GDM
B	>20 years…or…	….0-9 years	No	IDDM without EOD
C	10-19 years…or…	…10-19 years	No	
D	<10 years…or…	…>20 years	Vascular / BGR	
F	D.M. + Nep**h**ropathy			IDDM with EOD
R	D.M. + Proliferative **R**etinopathy			
H	D.M. + Ischemic **H**eart disease			
T	D.M. + Renal **T**ransplantation			

National Diabetes Data group classification (1979)

- Type 1: IDDM
- Type 2: NIDDM
- Type 3: DGM
- Type 4: IGT

❷ Confirmatory (GTT): glucose challenge

(Modified O'Sullivan test)

- Daily 150 gm CHO diet is allowed for 3 days (with no smoking ¤)
- FBS is determined (after overnight fasting of 8–14 hrs)
- Then give 50 (UK), 75 (WHO),100✓ (USA) gm glucose in 400 ml water
- Readings are taken hourly for the next 3 hrs & a curve is drawn

	Normal		Renal	Alimentary	D.M.
	Blood	*Plasma*			
FBS	<90	<105	All readings are as normal	N	At least 2 readings are > N
1-hr	<165	<190		> 180 mg %	
2-hr	<145	<165		N	
3-hr	<125	<145		N	
Urine sugar	*All are negative*		+ve only at peak	+ve at 1-hr peak	+ve at any time

* Single abnormal value is called → impaired glucose tolerance (IGT)
* Normal values are to be repeated at 3rd trimester in the *high-risk group* ✓
* If glucosuria found on 2 separate occasions during ANC → GTT ¤

❸ Investigations for control

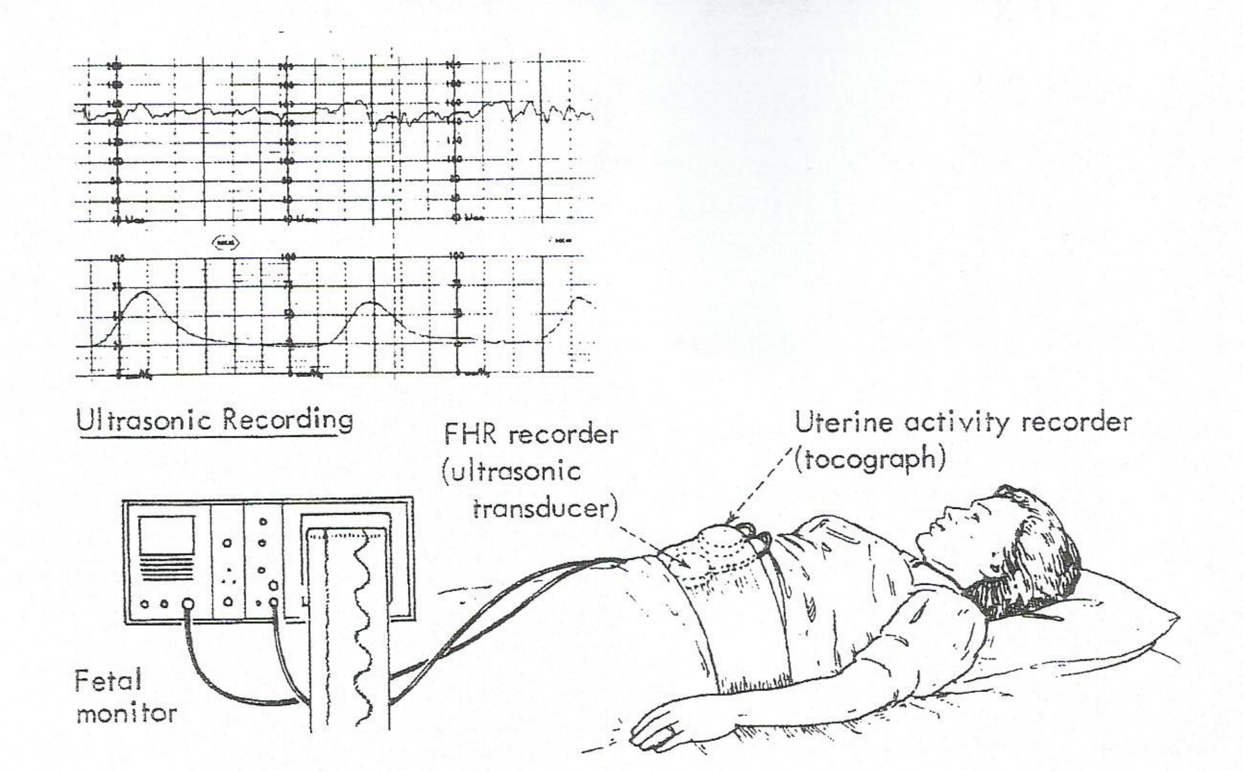
* Level of glycosylated Hb (HbA_{1c})
- This indicates the control over the previous 2–3 months (N = 5–8%)
- Above 10% → poor control
- Above 12% in early pregnancy → ↑ % of CFMF ¤

* Glycosylated serum proteins: fructosamine (reflects previous …… control)

❹ Investigations for complications

* On pregnancy
- Urine analysis → UTI
- Albuminuria → PET

* Of DM
- *Kidney function tests* to detect renal affection
- *Fundus examination*
  . Background retinopathy → not dangerous
  . Proliferative retinopathy → very serious & may lead to blindness → refer to laser therapy → if still progressive → consider TOP


Ultrasonic Recording
FHR recorder
(ultrasonic
transducer)
Uterine activity recorder
(tocograph)
Fetal
monitor

Management

❶ Preconceptional care

- **Postpone pregnancy**....till good DM control (as evidenced by HbA_{1c})
- **Advice against pregnancy if**
  - $HbA_{1c} > 12\%$ (high risk of CFMF) } *indications*
  - Marked renal affection is present } *of therapeutic*
  - Progressive proliferative retinopathy } *TOP*
- **Oral hypoglycemic drugs are not used**
  - ↘ they cross the placenta: CFMF + ↑ fetal hyperinsulinism

❷ Antenatal care

- **Time** → 2 wks (3 in GDM) till 32 wks, then weekly
- **Place** → a specialized antenatal clinic (obstetrician, physician, dietitian)
- **Aim** → * control of DM & prevention of its progression
  * early detection & management of comp. (general / obstetric: M&F)
- **Control 'STRICT'** ✓
  - ➡ **Diet**
    - Sufficient alone only in mild cases (GDM A_1, IGT)
      - . Give CHO (50% = 200-250 gm) fats (30%) proteins (20%) ¤
      - . Carbohydrates should not be in the sugar form (rapidly absorbed)
      - . Average 1800–2400 Kcal/d ± 300 Cal in 3rd trimester ¤
      - . Total calories are divided among 3 major meals + 3 snacks
    - Exercise allowed → physical activity should be moderated
  - ➡ **Diet + insulin**
    - Split schedule system (7 am & 5 pm)...regular + intermediate
    - Indication
      - . GDM A_1 if diet failed: FBS> 105...1hr PPS>140...2hr PPS>120
      - . GDM A_2, Class B–T
- **Investigations**
  - ➡ **Mat. comp :** Of DM → renal FT, liver FT, fundus, serial HbA_{1c}
    On preg → screen for PIH, infections (urine, vaginal C&S)
  - ➡ **Fetal surveillance**
    - GDM A_1
      - U/S at 38 weeks to exclude fetal macrosomia
      - CTG & BPP weekly starting from 34 weeks
    - GDM A_2 & IDDM B–T (pregestational IDDM)
      - U/S at . 18 – 20 wks (excludes CFMF)..........± MS-αFP
        . Serially (for macrosomia or IUGR)......± Doppler
      - CTG & BPP weekly starting from 32 weeks

③ Termination of pregnancy

➤ Time

- Diabetics should not be allowed to pass dates ⟹ >40 wks ✗
- *In mild cases under excellent control* (GDM class A_1) ⟹ 40 wks
- *Insulin requiring diabetics* (Class A_2, B, C, D)
  - Well controlled, no F/ M complications ⟹ 38–40 wks
  - Not well controlled: once document maturity ⟹ 37 wks
  - Earlier TOP < maturity if F/M distress occur ⟹ <37 wks
- In cases with repeated unexplained IUFD terminate ⟹ 1-2 earlier

> Before any elective termination
> ↘ Tests for fetal lung maturity should be done by *amniocentesis*:
> - Shake test (easy)
> - L/S ratio (more specific → widely used)
> - Phosphatidylglycerol → RDS may occur in spite of mature L/S

➤ Mode

- Cesarean section: ⊷+
  - * Macrosomia (> 4kg ?!). This is > in GDM
    - . Deposition of glycogen is more at shoulders & fetal liver
    - . The disproportion between fetal head / abdomen → sh. dystocia
  - * Previous history of unexplained IUFD
- Vaginal → by AROM ± syntocinon (?) + intrapartum fetal monitoring

➤ Insulin management during TOP

- *Keep maternal euglycemia* (80–100) → to avoid fetal hypoglycemia
- *Before labor* → stop morning insulin (taken *only* at bedtime)
- *During labor* → 500cc 5% glucose + 5 units crystalline insulin by drip /5 hrs
- *After labor* → insulin requirements usually drop immediately
  - If glucose level is > 200 → S.C. regular insulin when needed
  - If glucose is persistently > 200 → resume combined regular & NPH

④ Postpartum care

- Care of the newborn……at the NICU by expert pediatrician
- Breast-feeding….encouraged ¤ (lactation is anti-diabetogenic)
- Contraception
  - COC are contraindicated in those with vascular changes (∴use POP)
  - IUCD are contraindicated esp. in those with depressed immunity
    - ↘ Used with caution (e.g. vaginal discharge)
    - ↘ Threads cut short ± prophylactic antibiotic

Glucose Monitoring and Insulin Dosing During Pregnancy

Insulin Type and Dose Time	Time Impact Seen	Target Glucose Level (mg/dL)
Evening NPH	Fasting	70–90
Morning Humalog	Post breakfast	100–139
Morning NPH	Post lunch	100–139
Evening Humalog	Post dinner	100–139

Instruction for Adjusting Insulin Dosage

1. Establish a fasting glucose level between 70–90 mg/dL.
2. Only adjust one dosing level at a time.
3. Do not change any dosage by more than 20% per day.
4. Wait 24 h between dosage changes to evaluate the response.

Insulin therapy

➤ **Indications:**

. GDM A_1 if diet failed
. GDM A_2, Class B–T

➤ **Drugs used:**

. *Combination of* Crystalline (regular / short) } *better human*
+ NPH (isophane / intermediate) } *e.g. Mixtard*
. Long acting insulin (protamine zinc) ✗ are not used: poor control

➤ **Dosage**

- In class B–T → no change in previous dosage (if sugar is controlled)
- In GDM A_2 give → 0.6 u/kg (1st trimester), 0.7 u/kg (2nd), 0.8 u/kg (3rd)
- The calculated dose is then divided

Morning (7 AM)	Evening (5PM)
⅔ dose ↓ ⅓ crystalline + ⅔ NPH	⅓ dose ↓ ½ crystalline + ½ NPH

Assess action at		*Urine*
Morning	®	10 AM
	NPH	5 PM
Evening	®	8 PM
	NPH	7 AM

- Urine should be free of glucose: if present → increase the corresponding insulin dose (but gradually!)
- Gluco-meter (capillary blood estimation) is *better* ¤ used instead of urine for follow up of dosage (d.t. gluco.....)
- Check FBS & 1 hr-PPS after each meal until control, then twice weekly → then once weekly (1 hr-PPS should be < 140 mg/dl ¤)

➤ **In resistant cases**

. May give *three* times daily injections (NPH at 5 PM is given 1 hr < bed time snack → better control of nocturnal hypoglycemia & FBS)
. Continuous infusion pump (not superior on injections)

➤ **Site of injections**: given S.C. in abdomen ✓(?) → arms → thigh → buttocks
Patient is to be taught with variation of the injection site

➤ **The patient should be warned against symptoms of hypoglycemia**
(headache, sweating, palpitation, hunger, epigastric pain, dizziness) & if any of these occur she should take a readily available carbohydrate (candy or biscuit) + ↓ the corresponding insulin dose

KEY POINTS

1. Gestational diabetes occurs in 1% to 12% of pregnant women.
2. Risk factors for gestational diabetes include Hispanic, Asian American, Native American, and African American ethnicity, obesity, family history of diabetes, and prior pregnancy complicated by gestational diabetes, macrosomia, shoulder dystocia, or fetal death.
3. All pregnant women should be screened for diabetes between weeks 24 and 28. High-risk women should also be screened at their first prenatal visit.
4. Fetal complications of gestational diabetes include macrosomia, shoulder dystocia, and neonatal hypoglycemia.
5. Pregnancy management should include frequent health care visits, thorough patient education, American Diabetic Association diet, glucose monitoring, fetal monitoring, and insulin or an oral hypoglycemic agent as indicated.
6. Patients should generally be induced between 39 and 40 weeks gestation. Intrapartum insulin and dextrose are used to maintain tight control during delivery. Cesarean section is offered if fetal weight is over 4500 g.

KEY POINTS

1. Maternal complications of diabetes during pregnancy include hyperglycemia, hypoglycemia, urinary tract infection, worsening renal disease, hypertension, and retinopathy.
2. Fetal complications of diabetes during pregnancy include spontaneous abortion, congenital anomalies, macrosomia, IUGR, neonatal hypoglycemia, respiratory distress syndrome, and perinatal death.
3. Pregnancy management is optimized by a preconceptional visit, early prenatal care, thorough patient education, tight glucose monitoring and management with insulin, fetal monitoring, and thoughtful plan for delivery.
4. Motivated type 1 diabetics can usually maintain tighter control on an insulin pump. Management in labor and delivery usually requires an insulin drip; however, insulin requirements decrease dramatically postpartum.

Important points

Gestational DM

- CHO intolerance recognized for the 1st time during pregnancy & disappears after pregnancy (whether insulin is used or not for ttt)
- Screening for GDM should be performed between 24-28 wks

Types	Management	Termination
Low risk / A_1	diet control	Left till term (never past-dates)
High risk / A_2	diet + insulin	Managed as IDDM

- More liable to macrosomic fetus (IDDM → CFMF & IUGR)
- Postpartum Consequences
  - Risk of type II DM (**50%** may develop overt DM within 20 yrs) ¤
  - Recurrence of GDM (reported in ⅔ of cases – esp in obese women)

Glucose intolerance

- If there is only *one* abnormal value in the 3 hr-GTT
- These patients are still *at risk* for → macrosomia & PIH
- TTT → only diet control but *recheck* FBS & 1hr-PPS every 2 weeks

Q. Types of diabetic comas

- Diabetic ketoacidosis
- Hypoglycemic coma
- Hyperglycemic hyperosmolar non-ketotic
- Uremic & cerebrovascular strokes

Q. Types of insulin Beef / pork / *Human* ✓✓ (mixtard) better

	≈ Onset	≈ Peak	≈ Duration
Short acting (regular, semilente)	½ hour	3-4	6-8
Intermediate (NPH, lente)	2	8-12	16-24
Long acting (PZI, ultralente)	8	12-16	24-32

NPH is the neutral protamine of Hagerden

Q. Types of ketone bodies → Acetone, Aceto-acetic, β-hydroxy-byuteric acid

Somogyi phenomena

- Increased morning FBS + nightmares.
- Explained by nocturnal hypoglycemia followed by exaggerated counter-regulatory mechanisms → ↑ FBS
- TTT → ↓ NPH of 5-PM

Dawn phenomena

- Increased morning FBS + absent nocturnal hypoglycemia
- TTT → ↑ NPH of 5-PM



Heart disease with preg. (1%)

Types

- Rheumatic → 93% (MAT esp → MS)
- Congenital → 7% (> in developed countries)
- Others → 1% (e.g. IHD, arrhythmias, cardiomyopathy)

Classification

American New York Heart Association [NYHA] divided HD into 4 grades (according to the functional capacity of the heart):

CLASS I Organic heart disease but with ***no*** limitation of physical activity

CLASS II Some limitation of the ordinary activity in the form of dyspnea, fatigue, palpitation, pain. . A → ***mild*** limitation
. B → ***moderate*** limitation

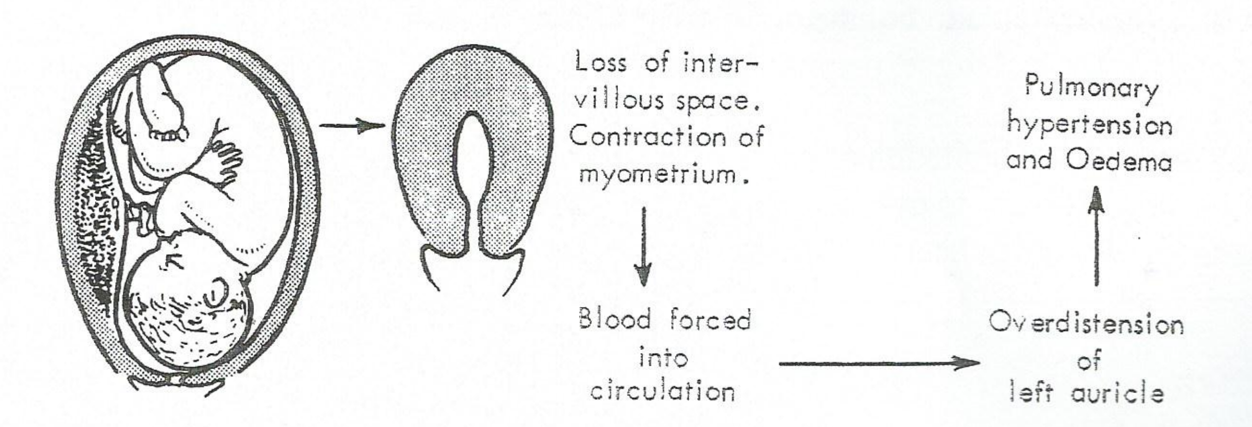
CLASS III ***Marked*** limitation of physical activity as it will lead to dyspnea on less than ordinary activity

CLASS IV They are in heart failure with dyspnea even ***at rest***

Clinical Picture

History

- Personal
  - Name / Age / Marital status / Parity
  - Address : --------- RHD is > in damp non-sunny area
  - Occupation : ----- may need advice against marked physical effort
  - Special habits :--- must stop smoking
- Complaint & HPI:
  1] *PVC* → **dyspnea**, orthopnea, PND, cough, expectoration, hemoptysis
  2] *SVC* → engorged neck veins, rt hypochondrial pain, ascites, LL **edema**
  3] *Rheumatic activity* → carditis, arthritis, chorea gravidarum, SC nod, erythema
  4] *IEC* → fever, symptoms of HF, CNS sympt., hypochondrial pain, haematuria
  5] *Arrhythmia* → **palpitation**
  6] *Cyanotic heart disease* → cyanosis (**malar flush** in pregnancy)
  7] Ischemia → anginal pain
- Menstrual history → for dating
- Obstetric history → previous HF in pregnancy
- Past history
  - Medical → rheumatic fever, duration of heart disease, attacks of failure
  - Surgical → valve replacement
  - Drugs → anti-failure / anti-coagulant



H.F. immediately after labor

Examination

- **Peripheral** ☞ *Normally we could* *see* ⇨
  - Signs of SVC
    - Neck veins → not reliable d.t. ↑ed blood volume
    - Edema → may occur due to (pregnancy or PET)
    - Enlarged liver → may be difficult to palpate due to large uterus
  - signs of hyper-dynamic circulation e.g. H_2O hummer pulse, cap. pulsation
- **Central** ☞ *Normally we could* *hear* ⇨
  - Splitting of the 1st sound
  - Appearance of the 3rd sound
  - Soft systolic murmurs (< 2/6)
  - Shift of apex beat from 5th to 4th intercostal space

Complications ΦΦ

Effects of pregnancy ➡ HD

1] *Deterioration by one clinical grade due to:*
   - * Blood volume ↑ 40-50%
   - * COP ↑ 30% (due to↑ blood volume + ↓ P.R.)
     = (Heart rate ↑ 10-15 b/m) X (Stroke volume ↑)

2] *Heart failure may occur in*
   - * **Pregnancy** → at 30–34 wks (max.↑ in COP & blood volume)
   - * **Labor** → pain + uterine contraction →↑ VR to heart → ↑COP
     (2nd stage > 1st stage d.t. more bearing down)
   - * **3rd stage** → return of the blood in the uteroplacental circulation (500-700) to G.circulation (after placental separation)

3] *RHD:* . IEC after any procedure (esp in puerperium)
   . Recurrence of rheumatic activity is rare (but serious if occurred)

4] *Liability to arrhythmia*

5] *Liability to more cyanosis* in cyanotic heart disease

6] *Liability to the thromboembolic* complications (due to ↑ stasis)

Effects of HD ➡ pregnancy

☆ Maternal
- Polyhdraminos (d.t. congestion)
- PTL (d.t. soft cervix)
- PPhge (hypoxia + ergometrine contraindicated)

☆ Fetal
- Abortion & CFMF } Low COP → chronic hypoxia
- IUGR & IUFD } esp in cyanotic heart disease

Some give Heparin all through

* Advantage	* Disadvantage
- Doesn't cross the placenta - Short acting (2-4 hrs) - Have antidote ➠ protamine sulfate slowly IV	- Overdose ➠ bleeding tendency - Long use .Thrombocytopenia .Osteoporosis (∴use calciparine)

Some give Oral anticoagulant all through as

- The risk of *over* or *under* control by heparin is more serious ¤ than the minimal recorded risk of fetal affection d.t. OAC
- FFP rapidly reverses action of OAC (if bleeding occurs)
- Also, there is antidote → Vit K (for both mother & fetus)
- NB:- ***Dindivan*** is contraindicated during lactation ¤

Investigations

1. X-ray ?! (+ abdominal shield) → cardiomegally
2. ECG / Echocardiography
3. Rheumatic fever → ESR, CRP, A-SOT

Treatment

❶ Preconceptional control

➡ Pregnancy is contraindicated in: ΦΦ

- Class III & IV
- Cyanotic heart disease as Eisenmenger syndrome
- Severe Aortic stenosis or 1ry P. hypertension (d.t. limited COP)
- History of - HF in previous pregnancy
  - Rheumatic activity / IEC in the past 2 years

➡ If they become pregnant, therapeutic TOP is better done in 1st trimester. After 14 wks inducing abortion is more *hazardous* ☠ than continuing pregnancy

❷ Antenatal care

Done in ⇨ a specialized antenatal clinic (obstetrician, cardiologist)
Done for ⇨ control of HD & early detection & management of comp
Done every ⇨ 2 wks till 32 wks, then weekly
Done by ⇨

- **Rest** → some hospitalize at [30–34 wks] then to plan labor [36–37 wks]
- **Diet** → salt restriction
- **Drugs** →
  - Avoid anemia / infections (esp. resp tract) → ppf to heart failure
  - Long acting (benzathine) penicillin 1.2 million IU/month ✓
  - Class III & IV → digitalis, diuretics, aminophylline
  - Valve ↗ replacement with pregnancy:-
    - ☆ Warfarin (5mg) or Phenindione (50mg)
    - ☆ BUT use Heparin (5000 IU/S.C./8hrs) during ⤵

1st trimester (as warfarin ↑ CFMF as microcephaly, optic atrophy, chondrodysplasia punctata + ↑ fetal hge)
2-3 weeks before delivery ⇨ shift back to heparin
At onset of labor ⇨ stop heparin
After labor (6-12 hrs) ⇨ give OAC + heparin (till OAC acts)
3 days later ⇨ stop heparin.....then continue only by OAC

Regimens for endocarditis prophylaxis during labor and delivery

Low risk regimen	Amoxicillin, 3 g p.o. 1 h before procedure or at onset of labor Repeat 1.5 g p.o q.6 h until after delivery
Standard regimen	Ampicillin, 2 g i.v. plus gentamicin, 1.5 mg/kg i.v. (do not exceed 80 mg) 30 min before procedure or at onset of labor. Repeat above q.8 h until after delivery
Penicillin-allergic standard regimen	Substitute vancomycin, 1 g i.v. over 1 h q.12 h for ampicillin

History

Etienne-Louis Arthur Fallot (1850–1911) was a professor of forensic medicine and hygiene in Marseille. He had a reputation as an astute clinician and for accurate careful physical examination.

❸ Termination

◈ Time

➡ Class I & II.......left for smooth spontaneous of labor (no induction?)
➡ Class III..........if completed here family → better to terminate
..........if insists on pregnancy → continue in hospital
➡ Class IV..........control the HF 1st medically, then terminate

◈ Route

▸ vaginal ✓✓

1st stage

- *Semi-sitting position* with no bearing down
- *Adequate analgesia*
    * Morphine (10mg) or Pethidine (100mg)
    * Epidural analgesia
- *Intermittent* O_2 + antifailure ttt if needed
- *Close observation*: [P-BP-Temp] + FHS + Uterine contraction
- *Prophylactic antibiotics* (GBS✓) & delay AROM as possible
    * 2 g ampicillin + gentamycin 1.5 mg/Kg
    * given 1 hour before placental separation
    * Ampicillin is repeated once after 8 hours

2nd stage

- *Usually easy* (small baby + soft cervix)
- *Shorten* 2nd stage & avoid bearing down by low forceps or ventouse

3rd stage

- Avoid ergot IV (↑ heart load due to VC + uterine contraction)
- Lasix may be given ✓ (if heart failure)
- Guard against PPhge (may give ¼ mg IM)

▸ cesarean section ➡ ⊷ +

1] *Aortic stenosis* (post-stenotic dilatation may rupture during bearing)
2] *1ry pulmonary hypertension*
3] *Eisenmenger syndrome*
4] *Marfan syndrome* (if having dissecting aortic aneurysm)

❹ Postpartum care

* *Breast-feeding allowed* except in severe cases (class III & IV)
* *Advise for contraception* ⇨ Mechanical methods or Sterilization
    - o IUCD → ascending infection (if used: aseptic technique + proph. abtc)
    - o COC → thrombosis (if used: POP)

SOME IMPORTANT CARDIAC CONDITIONS

1] Mitral Valve Prolapse

Pathology: Myxomatous degeneration of one or both of mitral valve leaflets → prolapse into left atrium during systole

C/P: - Asymptomatic mainly
- May → palpitation, dyspnea, chest pain, syncope

Treatment during labor: Controversial → only inderal

Most don't give antibiotics except if associated with MR

2] Peripartum Cardiomyopathy

Definition: Dilated cardiomyopathy → HF in 3rd trimester / puerperium

Incidence: 1/1.500 – 1/4.000

Etiology:
- Unknown
- May be → viral infection, autoimmune
- Pdf → PET, twins, genetic predisposition

Prognosis: (Fatal) . Mortality is 25–50%
. Mortality in next pregnancy is 80%

Treatment
1. **Digitalis**, hospitalization
2. ↓ afterload (Hydralazine), ↓ preload (diuretics)
3. **Heparin** (risk of thrombus formation in dilated heart)

3] Coarctation of Aorta

Definition
- Hypertension only in upper limbs
- Normal / low pressure in lower limbs
- May be confined only to left arm (coarctation of left subclavian)

Route of termination
- Vaginal delivery allowed
- C.S. only in other obstetric indications

4] Marfan Syndrome

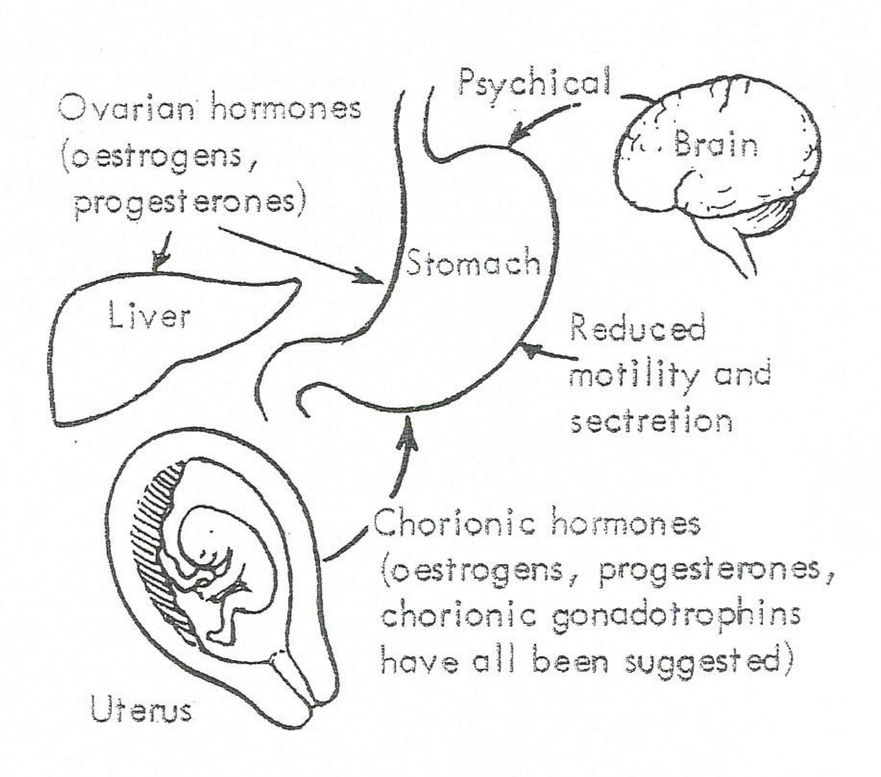
Etiology → defective CT

Clinical picture
- Mitral valve prolapse / incompetence
- Aortic dissection (intimal tear) → acute chest pain + shock

5] Surgery in pregnancy

Tight MS (< 1cm) Balloon catheterization may be done (2nd trimester)

Valve replacement is contraindicated (hear lung machine + anticoagulation)


Psychical
Ovarian hormones
(oestrogens,
progesterones)
Brain
Stomach
Liver
Reduced
motility and
sectretion
Chorionic hormones
(oestrogens, progesterones,
chorionic gonadotrophins
have all been suggested)
Uterus

Vomiting with preg.



① Emesis gravidarum (morning sickness)

➢ Definition
- N & V in the 1st trimester (max 6th – 12th week)
- It doesn't affect the general condition

➢ Incidence ⇨ very common (80%) esp in PG, esp in the morning

➢ Etiology ⇨ unknown

➢ Management
- Reassurance → it disappears spontaneously
- Small frequent meals →. Better dry CHO meals
  . Avoid immediate recumbency after meals
  . Fe therapy is temporarily stopped (nauseating)
- If not responding → antiemetics

② Hyperemesis gravidarum ✍

➢ Definition
- Severe vomiting to a degree that } pernicious
- Affects the general condition } vomiting of preg

➢ Incidence ⇨ 0.1–1 %

➢ Etiology ⇨ theories
- Psychological: as it
  . Start only after knowing that she is pregnant
  . Vomiting only infront of her husband ☹ → more in neurotic females
- Hormonal
  . ↑ HCG (as in V.mole & twins)
  . ↑ T_3, T_4 ....transient....no need for ttt
  . ↓ Corticosteroids
- Allergic → against CL of pregnancy, sex steroids
- Deficiency (esp Vit B_1 & B_6)

➢ Pathology ⇨ vomiting ⊃
- Starvation → dehydration → starvation ketosis & elect. imbalance
- Liver → fatty change & centrilobular necrosis
- Kidney → tubular necrosis
- Heart → brown atrophy
- Brain → petechial hge & congestion
- Retina → hge, optic neuritis, detachment

Clinical Picture

- Excessive vomiting (allover the day & not related to meals)
- Dehydration → . ↓BPr, ↑pulse, ↑temp……… oliguria, constipation
  . ↓weight, sunken eyes + jaundice, dry inelastic skin
- CNS → . Peripheral neuritis
  . Wernicke's encephalopathy – d.t. Vit B_1 def

Investigations

- Diagnosis ⇨ **Urine analysis** → Ketone bodies✓ + no glucose
  ⇨ **CBC** → ↑ Hct…..↓ (Na, K, cl)
- Etiology ⇨ **U/S** (Twins, V.M.) + T_3, T_4
- Complication ⇨ **Fundus**, **Liver + Renal function tests**

Treatment

[1] **Hospitalization** ➡ reassurance (& isolation!)

[2] **Diet**

- NPO + IV fluids (till 48 hrs after vomiting stops)
- Then restart gradually by clear liquids + CHO meals (no fats, spices)
- If failed → TPN + thiamine (B_1)

[3] **Drugs**

- Sedatives → phenothiazines (chlorpromazine)
- Antihistaminic → promethazine (phenergan)
- Antiemetics → . metoclopramide (primperan), Cortigen B_6
  . navodoxine, motilium (domperidone)
- In resistant cases → Zofran (ondansteron: 5–HT blocker) ± steroids

[4] **Observation for**

- Vomiting: → frequency
- Vital data: → BPr, P, T
- Urine analysis → daily
- Organ function tests + Fundus → weekly

[5] TOP

** *Indication*

Deterioration of general condition in spite of ttt [P >100, T > 38]
Deterioration of organ affection [renal / hepatic / CNS / retina]

** *Methods*

< 14 weeks → Suction evacuation or D&C
> 14 weeks → may end in HYSTEROTOMY …….why?

Complications of cholestasis of pregnancy

Complications	Comments
PPH	Due to malabsorption of vitamin K
Intrauterine death	2–4%, with risk increasing with gestation
Premature labor	40% will deliver before 37 weeks
Fetal distress in labor	Meconium-stained amniotic fluid likely
Fetal and neonatal intracranial hemorrhage	Due to maternal absorption of vitamin K

---- Extras ----

➤ DD of vomiting in pregnancy Φ

1. Morning sickness (emesis gravidarum)
2. Disturbed ectopic pregnancy
3. Vesicular mole
4. ....Preeclampsia (severe)
5. ....Pyelonephritis
6. ....Polyhydramnios
7. Gyn. *conditions* → twisted ov. swelling, red degen. of fibroid
8. Medical *conditions* → food poisoning, hepatitis
9. Surgical *conditions* → appendicitis, cholecystitis, peptic ulcer

➤ DD of Jaundice in Pregnancy Φ

Pregnancy induced	Pregnancy associated
- PIH & HELLP syndrome	- Hemolytic J.
- Severe hyperemesis gravidarum	- Obstructive J.
- Intrahepatic cholestasis of preg	- Hepatocellular (**V.H.** ✓✓)
- Acute fatty liver of pregnancy	- Drugs

➤ Intrahepatic cholestasis of pregnancy

**Incidence* → the commonest liver disorder UNIQUE to preg. (esp. in 3rd trim)

**C/P* → mild Jaundice, pruritus + ↑ in Bile acids are DIAGNOSTIC

**Comp.* → PTL & IUFD

**Investig.* → . ↑ed (alkaline phosphatase, Bilirubin –direct–, SGOT & SGPT)
. Prolonged prothrombin time

**Treatment*

- mother . cholestyramine 4gm 1x4 ± Phenobarbitone for pruritus
  . Vit K 10 mg to improve the PT
  . Corticosteroids may help
- fetus → TOP once mature → MARKED improvement within 2 days

**Prognosis* → avoid use of COC to avoid recurrence (70%)

➤ Acute fatty liver of pregnancy (rare)

**C/P* - symptoms like viral hepatitis → fever, N&V, Jaundice, upper abdominal pain
- Then → symptoms similar to PET → then acute LCF

**Complications* → high MMR, PNMR

**Treatment* → TOP + liver support

Urinary tract dis. in preg.

[1] Asymptomatic (silent) Bacteriuria 

Definition (4–7%)

- Presence of >100.000 organisms of a single colony /ml urine
- In absence of any *symptoms* or *pus* cells

Effects on pregnancy → ↑ed liability to:

- Acute Pyelonephritis (in 25% of cases)
- Anemia
- PIH & IUGR
- PROM & PTL

Diagnosis

- No symptoms ¤ ∴ screening tests MUST BE DONE in 1st visit ⤸
  Colony count (by clean catch technique: MSU)

Treatment

1- Broad spectrum antibiotic (of high urinary concentrations) for 7–10 d
   [Ampicillin / Cephalosporins / Nitrofurantoin]
2- If failed → antibiotic according to C&S
3- Urine C&S is then repeated each trimester ¤

[2] Acute Pyelitis & Pyelonephritis

Definition (1%) → acute infection of renal pelvis & interstitial tissues

Predisposing factors ¤

- Short urethra
- Asymptomatic bacteriuria ⇨ ↑ % of infection
- Atony of the ureter (Pr. effect) ⇨ ascending infection
- Pressure on ureter (esp. Rt.) at pelvic brim ⇨ after 2nd trimester
- Catheterization (urinary tract is ……. until ……..) ⇨ after labor

Bacteriology

➤ **Organisms**

- E-coli → the most common ✓ (80%)¤
- Others → Gram +ve : staph / strept } or Mixed
  Gram –ve : Klebsiella / Proteus } Infections

➤ **Route of infection**

- *Ascending* infection……along the lumen or periureteric lymphatics✓
- *Lymphatic* spread……..from neighboring colon
- *Blood borne* from……..a septic focus (rare)

Clinical picture

* **Symptoms**
  - *General* → FAHMR + vomiting (sudden onset)
  - *Local* → severe loin pain, dysuria, haematuria / pyuria, frequency
* **Signs**
  - *General* → high fever, tachycardia
  - *local* → tenderness & rigidity in loins

Investigations

- Urine analysis → - Pus cells, RBCs
  - Acidic with fishy odour in E-coli
  - Urine C&S (esp in recurrent cases)
- Renal function tests → urea & creatinine *may* be affected
- Blood picture → ↑ ESR, TLC, CRP

Differential diagnosis ⇨ *acute pain in pregnancy*

Complications

- Chronicity..........Pyonephrosis..........Perinephric abscess
- Uremia..............Septicemia.............Septic shock
- PROM & PTL
- Recurrence in next pregnancy (20%)

Treatment

↪ **General**

- Hospitalization
- Ample fluids (oral or IV) + Analgesics + Antipyretics

↪ **Antibiotics** ➡ at least for 2 wks (best according to C&S)

- *Most commonly used* → . Ampicillin 500 mg 1x4
  . Cephalosporins 500-1000 mg 1x4
  . Nitrofurantoin 100 mg 1x4
- *If no response within 72 hrs* → repeat C&S
- *In resistant cases* → postpartum IVP, U/S → may reveal stones, congenital anomalies (esp if there is hematuria, hypertension)

↪ **Surgery**

- If ureter is blocked → ureteric catheter (pig-tail)
- If failed → nephrostomy

↪ **TOP** ⇨ If treatment fails to control the condition

[3] Acute renal failure in preg

> Definition → rapidly progressive azotemia

> Etiology

- *Pre-renal failure* (hypovolemia: accidental hge or hyperemesis Gr.)
- *Renal*
  - Sepsis (e.g. septic abortion)
  - PET, HELLP syndrome, DIC
- *Hepato-renal* → acute fatty liver in pregnancy

> Two types

- ACUTE TUBULAR NECROSIS (reversible)
- BILATERAL CORTICAL NECROSIS (rare & more worse)

[4] Chronic renal dis. with preg

> Effect of pregnancy ⇒ renal disease

- *Mild* (creatinine < 1.4 mg%)..............no change in renal function
- *Moderate* (creatinine 1.4 – 2.5 mg%)···.deterioration of renal function
- *Severely* (creatinine > 2.5 mg%).........usually don't get pregnant

> Effect of renal disease ⇒ pregnancy

- Effects
  - Superimposed PE ✓ (the most serious) ± accidental haemorrhage
  - Chronic anemia
  - Abortion / IUGR / PTL / IUFD
- Prognosis depends on
  - Development of *hypertension*
  - Development of *proteinuria*
  - Degree of *renal impairment*
  - *Type* of renal disease (diffuse & proliferative GN are worse)

> Treatment

- Mild ⇒ <u>*conserve*</u> under strict observation
  - ↳ monitor renal function + antihypertensives
- Severe ⇒ <u>*terminate*</u> or if deteriorating
  - ↳ advise sterilization or do it with termination

<u>NB</u>

- ☆ Pregnancy is possible on <u>*dialysis*</u> (but chronic anemia is the major problem)
- ☆ Pregnancy is possible after <u>*renal transplantation*</u>
- ☆ Delivery is better vaginal (even after renal transplantation, as kid. is in.......)

Anemia with pregnancy 

Definition ⇒ ⇩ in the amount of circulating haemoglobin (N= **12-16** gm%)

Normal changes in Hb in pregnancy

- PHYSIOLOGICAL ANEMIA (haemodilution) due to
  - RBC volume ↑ by 20–30 % but
  - Plasma volume ↑ by 40–50 %
- Lower limit is **11** gm/dl (Hct < 33%). Below this → PATHOLOGICAL ANEMIA ¤

Classification:

1] Dyshaemopoietic	1) **Iron deficiency anemia** ✓ **(the commonest dis)** ¤ 2) **Megaloblastic anemia** . Folic acid ↓ **(common)** ¤ . Vit. B_{12} ↓ **(rare)** ¤
2] Aplastic	**Bone marrow failure by** . Infections (viruses) . Drugs (chloramphenicol) . Malignancy (leukemia)
3] Haemolytic	1) **Intra-capsular** . Hb → haemoglobinopathies . Enzyme → G_6PD . Membrane → spherocytosis 2) **Extra-capsular → infection, drugs, hypersplenism**
4] Haemorrhagic	Acute (APHge, PPHge) or Chronic (e.g. piles)

- Microcytic (MCV < 80 fL) → iron def anemia, thalassemia
- Normocytic (MCV 80–100 fL) → sickle cell anemia
- Macrocytic (MCV > 100 fL) → vit B_{12}, folate def

Complications:

↪ Effect of pregnancy → anemia (worsened; d.t. ↑ Fe & vitamin demand)

↪ Effect of anemia → pregnancy

> Maternal

Pregnancy	- Abortion / Preterm labor - Antepartum hge.
Labor	- PPhge: atonic
Puerperium	- P. sepsis / Subinvolution - Defective lactation

> Fetal

The fetus obtains all its needs of iron & vitamins from the mother by active transport (even if she is anemic). However, there may be → . PTL & RDS

. ↑ PNMR (d.t. the maternal comp.)

Blood changes in pregnancy

	Non-pregnant	Pregnant
Haemoglobin (g/dl)	12–14	10–12
Red cell count ($\times 10^{12}$/l)	4.2	3.7
Haematocrit (venous)	40%	34%
MCV (fl)	75–99	80–103
MCH (pg)	27–31	No change
MCHC (g/dl)	32–36	No change
White cell count ($\times 10^{9}$/l)	4–11	9–15
Platelets ($\times 10^{9}$/l)	140–440	100–440
ESR (mm/h)	<10	30–100

ESR, erythrocyte sedimentation rate; MCH, mean corpuscular haemoglobin: MCHC, mean corpuscular haemoglobin concentration; MCV, mean corpuscular volume.

The requirements of elemental iron during pregnancy

Fetus and placenta	500 mg
Red cell increment	500 mg
Postpartum blood loss and 6 months' lactation	360 mg
Total	1360 mg
Saving from amenorrhoea approximately	360 mg
Net increased demand approximately	**1 gram**

① Iron Def. Anemia 90% ✓✓

▸ **Pathogenesis**

* Iron requirements in pregnancy are > iron absorption (inspite of its↑)
* Therefore iron stores in the mother are used to correct the difference
* If iron stores are already depleted or the mother is anemic
 ↘ iron deficiency anemia *occurs* or is *aggravated*

▸ **Normal Fe absorption**

- Daily absorption → 10% –↑ed to 20% in preg– of ferous supplied (10 mg /d)
 ↘ Non- preg. (1-2 mg), early preg. (2.5 mg), late preg. (6.5 mg)
- One gram is needed for the whole pregnancy

▸ **Etiology (Pdf)** - ⇩ nutritional intake or ⇩ stores
 - ⇧ loss → hge....vomiting....piles....parasitic infestations

▸ **C/P** ⇨ *Symptoms* → easy fatigability, dyspnea, palpitation
 Signs → pallor (lips, nails), tachycardia, glossitis, koilonychia, splenomegaly

▸ **Investigations**

- Hb % → < 11 gm/dl (<10.5 mg % recently)
- Blood picture → hypochromic microcytic, ↓ed (MCV, MCH, MCHC: <30 g/dl)
- Iron studies:
  - S. ferritin ↓ (reflects BM stores) <10 ng/ml (1st abnormal test✓)
  - Serum iron ↓ (N. = 60–180 μg/dl)
  - Bone marrow stores ↓
  - Total iron binding capacity ↑ (reflects ↓ transferrin saturation by 15%)

▸ **Treatment**

◆ *Prophylactic*
- Eradicate any pdf
- Improve diet + iron supplementation orally (after 1st trimester: N&V)
 ↘ Iron sulfate / gluconate / fumarate (30–60 mg/day)

◆ *Active*

1] Oral iron 1x3: during or after meals to supply 120–240 mg /day
 If → - Oral therapy fails (< 0.3–1 gm rise of Hb / wk)
 - Side effect: N&V, constipation occurred
 - Rapid results are needed, severe cases. Shift to ⤸

2] Parenteral iron
 Iron dextran (Imferon) IV or IM ✗
 Iron sorbitol (jectofer, IM) or iron sucrose (cosmofer, IV) ✓
 ** Side effects: Allergic + . IM → painful + sterile abscess
 . IV → thrombophlebitis

3] If severe anemia → Packed RBCs transfusion

② Megaloblastic anemia

- DNA replication is affected → ↓ nuclear maturation → affection of the 3 cell lines → *anemia*, *leukopenia* (infections), *thrombocytopenia* (bl. tendency)

➤ Folic acid deficiency ✓✓

Etiology → ↑ demand e.g. preg + ↓ intake (no vegetables + ↑cooking)

Effect → anemia + neural tube defects, cleft lip & palate (fetus)

Investigations

- Hb % < 11 gm/dl
- CBC → macr. hyperchr. (MCV >100fL), hypersegmented neutrophils ¤
- Folic acid level (N = 6-12 μg/L)
- Bone marrow → megaloblasts

Treatment

- Prophylactic → . Good diet ± folic acid supplements (800 μg/d)
  . Given also for → hemolytic anemia, anticonvulsant therapy
- Active → 5 mg/day + vit.B_{12} + **Fe** (to avoid unmasking ass Fe↓ anemia)

➤ Pernicious (addisonian) anemia (vit B_{12} def)

Etiology → long term vegetarian diet ¤ + intrinsic factor deficiency

Rare to become pregnant (usually infertile)

Effect → anemia + atrophic gastritis + neurological symptoms

Treatment → parenteral vit B_{12} cyanocobalamin (1000 μg/3 months/IM/ life)

③ Haemolytic anemia

➤ Thalassemia

1- Thalassemia minor → ↓ α chain synthesis → v. mild anemia → min. effect on preg

2- Thalassemia major → ↓ β chain synthesis → severe anemia, rare to become preg

TTT - Repeated blood transfusions + folic acid + **No** iron
- Splenectomy.....Desferal (iron chelating) to avoid haemosiderosis ✗

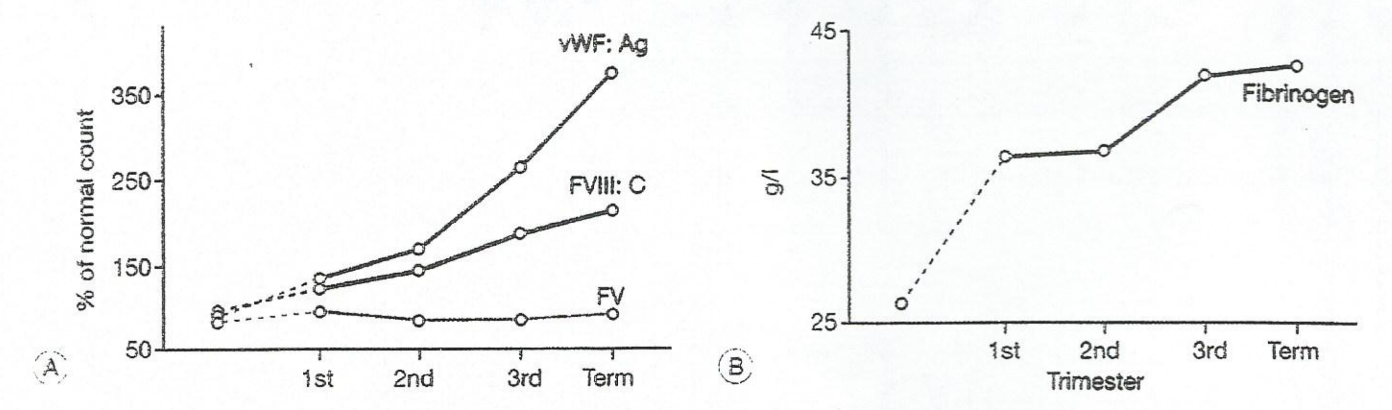
➤ Sickle cell anemia

1- Sickle cell trait → v. mild anemia → min. effect on preg (UTI ¤)

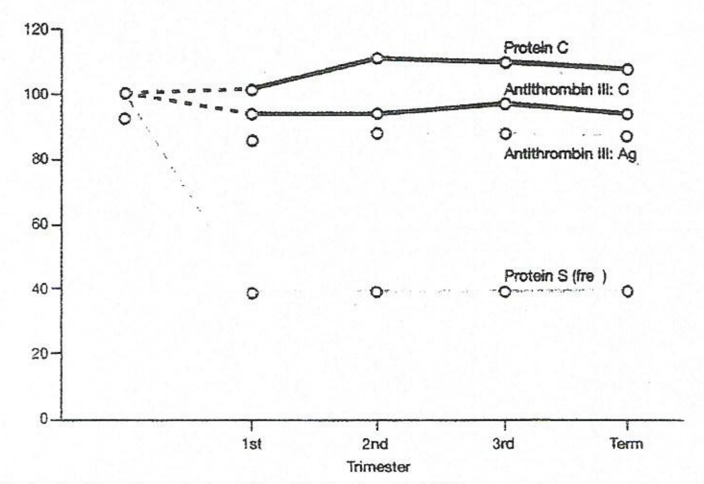
2- Sickle cell disease →

- Occlusive crisis: obstruction of vessels → infarctions
- Hemolytic crisis: anemia & jaundice

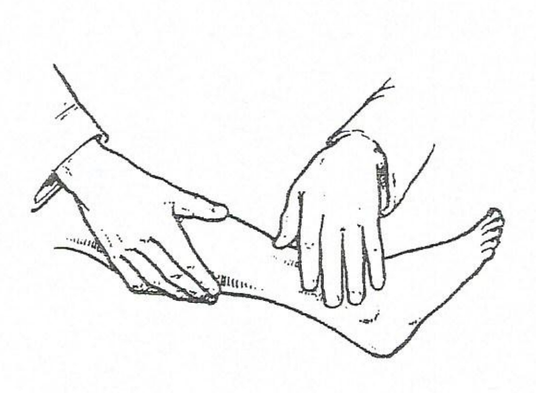
TTT - Prevent occlusive crisis by . Good hydration
. Avoid hypoxia & infections
- Repeated blood / exchange transfusion to ↓ level of HbS



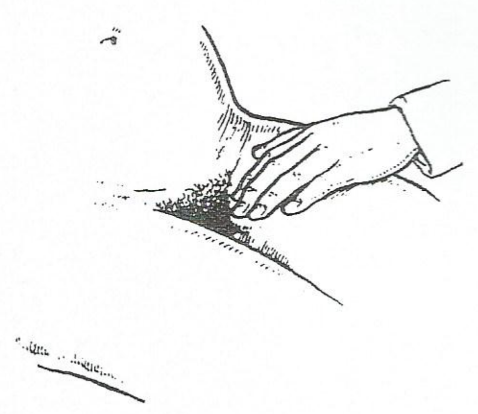
The levels of the procoagulants (A) factor VIII, von Willebrand factor and (B) fibrinogen rise in pregnancy. FV, factor V.



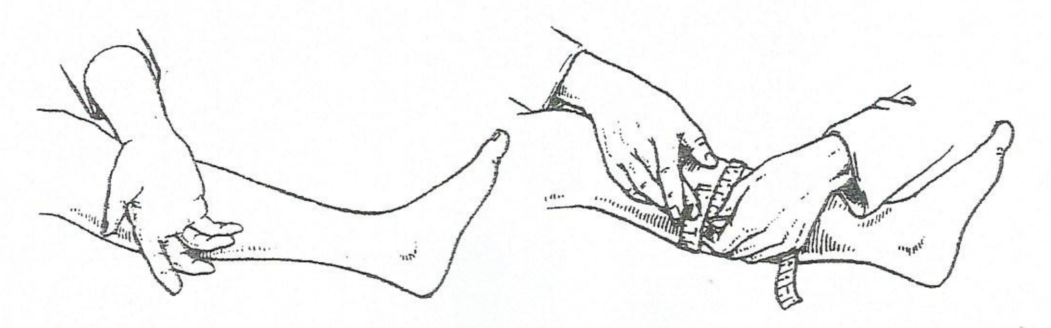
The levels of the anticoagulants antithrombin III and protein S fall in pregnancy.



Palpation of the calf demonstrates tenderness and oedema.



The femoral vein must also be palpated in the groin.



The affected leg may feel warmer to the back of the hand.

Careful measurement may reveal some swelling compared with the other leg.

Thromboembolic Diseases



➤ <u>Incidence</u> ⇨ 0.5–1%

Incidence is	→ equal in both ante- & post-partum periods
Untreated DVT	→ PE in 25% of cases with MMR 15%
Treated DVT	→ PE in 5% of cases with MMR 1%

➤ <u>Factors increasing risk of TED in pregnancy include</u> ↙

- **Mother**......................↑ed age & parity, obesity, ABO other than 'O'
- **Delivery**..............................operative > vaginal delivery (why??)
- **Previous thromboembolism**.........................recurrence risk is 15%
- **Thrombophilia**
  - Factor V Leiden deficiency → ↓ antithrombin III } recurrent
  - Protein C & S deficiency } TED &
  - Anti-phospholipid syndrome } fetal loss
- **Changes in Wirchow triad**
  - Prolonged immobilization
  - Congestive heart failure, dehydration, sickle cell disease

<u>Diagnosis</u>

☆ *Symptoms*

- DVT → acute painful swollen leg
  - . More common in the left LL (why..?)
  - . More common in ilio-femoral (more dangerous > calf)
- PE → sudden severe chest pain, dyspnea, cyanosis

☆ *Signs*

- Homan's sign (painful dorsiflexion of the ankle) } 30%
- Tender hard cord-like may be palpable } false +ve

☆ *Investigations*

- *Doppler & colored Doppler*
  - They are slightly less accurate in <u>pelvic</u> DVT (MRI may be used)
  - Venography still has better results in <u>calf</u> DVT
- *If PE is obvious* → start heparin immediately
  - No need for chest X-ray, ECG, blood gases
  - To confirm PE → perfusion (V/Q) scan, CT, MRI

Complications

Maternal: PE	DVT	Fetal	of ttt
MMR =15%. However, if recovery occurred → is usually complete	Post-phlebitic syndrome → valve destruction: edema, skin ulceration	APS may lead to habitual abortion (*important*)	On both 1- Mother 2- Fetus

Management

	Heparin	Oral anticoagulant ¤
Mechanism	activates AT_{III} → ↓ II & 9–12	Vit. K antagonist → ↓ II & 7–10
Curative Control	40.000 u/day IV, for 10 days *double APTT*	- Better for artificial valves - In VTE, it is used for 4-6 wks postpartum - PT is kept 2.5 – 3 x (INR) = international normalized ratio
Prophylaxis Control	10.000 units SC, twice daily *normal APTT*	
Contraindicatior	.Active bleeding, active ulcer . CNS aneurysms . Uncontrolled hypertension	Not used in the 1st trimester as it crosses the placenta ↝ fetal warfarin syndrome
Complications	- Haemorrhage - Thrombocytopenia - Osteoporosis (if used > 3m)	- Embryopathy - Chondrodysplasia Punctate - Fetal Hge: ICHge APHge

Low molecular weight (fractionated) heparin (LMWH)

- Fractionated heparin e.g. Enoxaparin (Clexane) ✓✓, Dalteparin (Fragmin)
- Dose → 1 mg /kg /12 hrs (Prophylaxis = 30–40 mg /12 hrs)
- Follow up → anti–Xa ✓ (not PTT)

	Fractionated heparin	Unfractionated heparin
Mode of action ATIII	Mainly X	Mainly II
Molecular weight (KD)	4.000 – 6.500	5.000 – 30.000
Bioavailability	90%	30%
Half life	4 hrs (SC or IV)	3 hr (SC)...1 hr (IV)
Side effects (hge, thrombo-cytopenia, osteopr.)	All are less with LMWH ¤ The main disadvantage is their high	

Thromboembolic disease

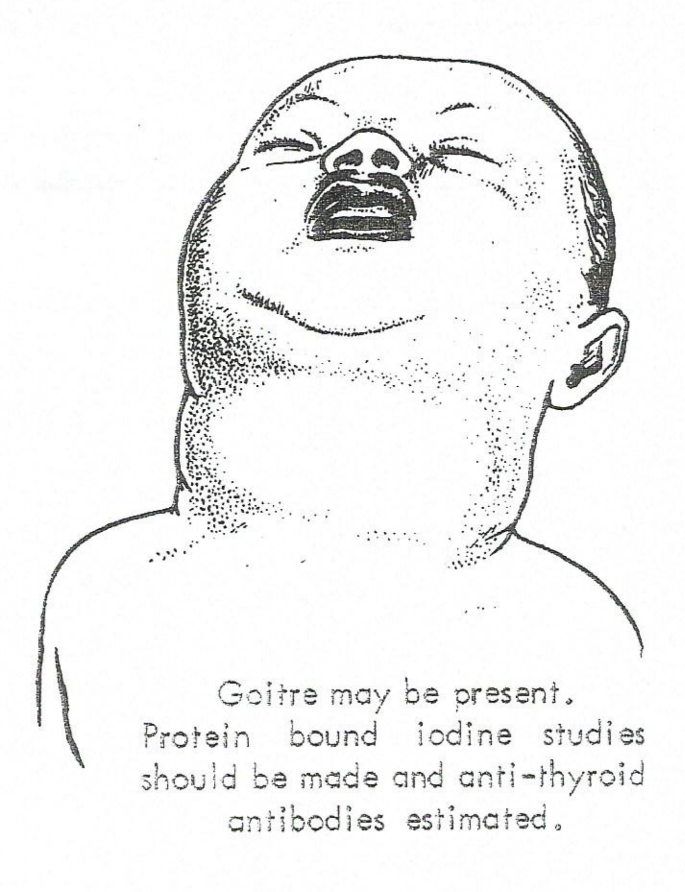
Risk factors for venous thromboembolism in pregnancy and the puerperium after vaginal delivery

Pre-existing	New onset or transient
Previous VTE	Surgical procedure in pregnancy or puerperium, e.g. ERPC
Thrombophilia Congenital Antithrombin deficiency Protein C deficiency Protein S deficiency Factor V Leiden Prothrombin gene variant Acquired (antiphospholipid syndrome) Lupus anticoagulant Anticardiolipin antibodies	Hyperemesis Dehydration Ovarian hyperstimulation syndrome Severe infection e.g. pyelonephritis Immobility (>4 days bed rest) Pre-eclampsia Excessive blood loss Long haul travel Prolonged labour Midcavity instrumental delivery
Age >35 years Obesity (BMI >30 kg/m^2) Parity >4 Gross varicose veins Paraplegia Sickle cell disease Inflammatory disorders, e.g. UC/Crohns Some medical disorders, e.g. nephrotic synd Myeloproliferative disorders, e.g. ET, PRV	Immobility after delivery

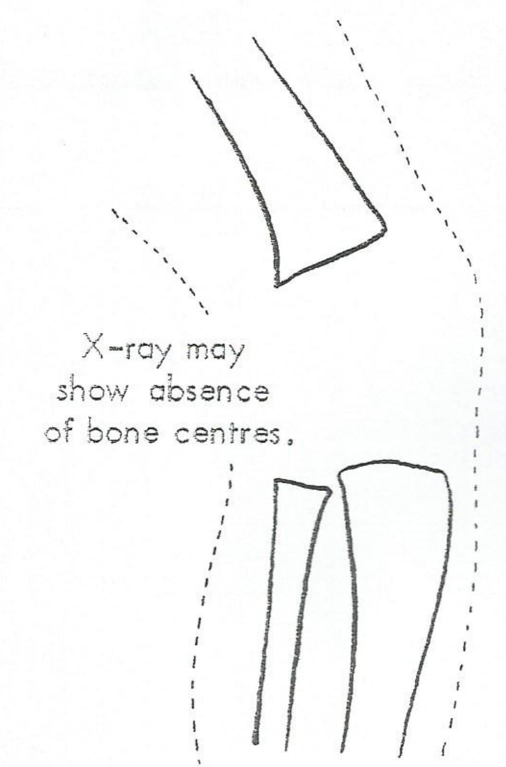
Thromboembolic disease in pregnancy – points to remember

- PTE is the commonest direct cause of death in pregnancy and the puerperium in the UK.
- Pregnancy is associated with an increased risk of thrombosis.
- The risk of DVT and PTE in pregnancy increases with increasing maternal age and obesity.
- Emergency caesarean section is associated with a greater than 20-fold increase of dying from PTE compared to spontaneous vaginal delivery.
- Objective diagnosis of DVT and PTE is vital.
- Treatment of VTE in pregnancy necessitates larger doses of LMWH and warfarin is avoided.
- Following acute VTE in pregnancy, LMWH must be continued for the rest of the pregnancy and the puerperium.
- Decisions regarding thromboprophylaxis in pregnancy relate to past history of VTE, the presence of detectable thrombophilia and the other identifiable risk factors.
- Women at high risk of recurrent VTE should receive antenatal and postnatal thromboprophylaxis with LMWH.
- LMWH and warfarin are safe to use in lactating mothers.

The baby should be examined carefully after birth.



Goitre may be present.
Protein bound iodine studies should be made and anti-thyroid antibodies estimated.



X-ray may show absence of bone centres.

Thyroid with preg 0.2 %



❶ Thyrotoxicosis

➤ **Complications**

↪ **Effect on thyroid**

- Usually tolerable course during pregnancy
- Condition may improve & exacerbates after delivery

↪ **Effect on Pregnancy**

✧ **Severe hyperthyroidism**: usually → anovulation → amen. & infertility

✧ **Maternal**

- Spontaneous abortion & PTL
- PIH & Congestive HF
- Hyperemesis gravidarum

✧ **Fetal**

- IUGR, IUFD
- Fetal tachycardia, neonatal hyperthyroidism
- Fetal thyrotoxicosis & goiter d.t. passage of autoantibodies (IgG)

➤ **Treatment**

↪ **Antithyroid drugs** ¤

- Propylthiouracil (drug of choice) ✓→ 200-400 mg /d
- Methimazole (carbimazole) → 20-40 mg /d
  - They cross placenta → fetal hypothyroidism & goiter
  - They are not an absolute contraindication to breast-feeding

↪ **Beta-blocking Agents**

- Propranolol (Inderal) 10 mg 1x3
  . Block the beta-adrenergic receptors
  . Prevent adrenergic effects of thyrotoxicosis
  . Block the conversion of $T_4 \rightarrow T_3$
- The aim ¤ → maintain the lowest possible doses of anti-thyroid drugs

↪ **Surgery:** Subtotal thyroidectomy is rarely indicated ¤ except:

- Failed medical ttt
- Cannot tolerate medical ttt
- Large goiters with significant tracheal obstruction

It does not eliminate the risk of transplacental passage of LATS and the possibility of fetal & neonatal thyrotoxicosis

↪ **Radioactive** iodine ablation (I^{131}) → ✗ contraindicated in pregnancy ¤

❷ Hypothyroidism

➤ Etiology

- o **Primary hypothyroidism:** (TSH is high)
  - o Hashimoto's thyroiditis (autoimmune) ✓✓
  - o Iatrogenic (Radioactive-iodine 131, surgery, antithyroid drugs)
  - o Iodine deficiency
- o **Secondary hypothyroidism:** (TSH is low) Rare, 2^{ry} to:
  - o Hypothalamic or pituitary disease, as in Sheehan syndrome?!
  - o Chromophobe adenoma of pituitary gland

➤ Complications

* **Maternal** → abortion, PIH, & abruptio placentae, heart failure ¤ ¤

* **Fetal** →. IUGR & IUFD

. Congenital hypothyroidism (→ obstructed labor) occurs in:

- RAI therapy for thyrotoxicosis ✓
- Rarely in hypothyroidism

➤ Investigations

- Low serum T_3RU
- ↑ Thyroid antibodies (antimicrosomal, antithyroglobulin) in Hashimoto
- TSH is low in 2^{ry} hypothyroidism

➤ Treatment

Replacement therapy

- L-thyroxine (T_4) 0.05–0.10 mg /day converted in body to T_3
- Breast-feeding is not contraindicated

Thyroid function in pregnancy

- o Increased due to
  - $\uparrow^{ed}$ production of TSH
  - $\uparrow^{ed}$ production of thyrotropin by the placenta
  - The thyroid stimulating effect of β-HCG
- o Leading to → ↑ total serum T_3 & T_4 & ↓ T_3RU
  However →TBG is also $\uparrow^{ed}$
- o Thus →
  - Free T_3 & T_4 remain normal
  - TSH remain normal



Respiratory disorders

❶ Breathlessness (dyspnea)

- **Physiological** → 50% of normal preg (*mechanical & prog* effect)
- **Pathological** → don't forget pulmonary embolism (acute dyspnea)

❷ Maternal Smoking

- **Tobacco smoke** (3800 constituents)
  - *Nicotine* → vasoconstriction
  - *CO* → combines with fetal Hb → carboxyhemoglobin → fetal hypoxia
  - *Benzopyrene* → mutagenic & carcinogenic

- **Effect of smoking on pregnancy**
  - IUGR ✓ Neonates are ± 200 g lighter than non-smokers
    Effect is dose related (number of cigarettes/day)
  - ↑ed PNMR → including sudden infant death syndrome
  - Spontaneous → abortion, PTL, PROM
  - APHge → placental abruption, placenta previa

❸ Bronchial Asthma (1%)

- **Complications**
  - **Effect of pregnancy ⇔ asthma:** no effect on frequency or severity
  - **Effect of asthma ⇔ pregnancy:** HTN, IUGR, PTL

- **Management**
  - **Regular medications are** CONTINUED (not teratogenic)
  - **Inhalation is better than oral agents**
    . Glucocorticoids (Betamethasone), Disodium cromoglycate & ipratropium ¤
    . β_2 agonists, Theophyllines (aminophylline) ¤
  - **Asthma** exacerbation **is not an indication** for elective delivery
  - **Hydrocortisone** is given during labor (300 mg /12 hrs)
  - **Avoid the following drugs** . Prostaglandin $F_{2\alpha}$ & E_2 analogues (misoprostol)
    . Methergine, Pethidine (not a problem in practice)

Surgery in pregnancy

	❶ Acute Appendicitis ¤	❷ Acute cholecystitis	❸ Ovarian torsion or Ruptured CL
Incidence	Commonest 1/1500 ✔ ¤	2nd common 1 /4000	uncommon
Pdf	Unknown	Relaxation of gall bladder (d.t. progest.)	Induction of ovulation
Diagnosis	difficult ¤	easier	↑ level of suspicion
C/P	Pain m.b. somewhat upwards. This depends on gest. age	Same like the non-pregnant	Acute unilateral pain ± N&V ± Adenexal swelling
Invest.	TLC is normally ↑ed in preg	Bilirubin, amylase Upper abd. U/S	U/S is essential for all (for differentiation)
Complicat.	Rupture, perforation, peritonitis..........fetus → abortion, PTL		
Treatment	Laparotomy (its site depends on gest. age) e.g. Ratherford extension	* Medical ttt (Fluids, NPO, A, A, A) * If failed (25%) or complicated → surgery	Look Cancer Ovary

Surgery during pregnancy ✔

Best time is at ....mid-trimester (why?)

Before surgery........document fetal life ± CTG

During surgery........tocolytic infusion, minimal uterine manipulations

After surgery

- Antibiotics
- Tocolytics, Profenid suppository ± progesterone
- Reassess fetal viability

Pain in pregnancy

❶	Pregnancy	
	☆ Early	Abortion, ectopic, incarcerated gravid RVF uterus
	☆ Late	Accidental hge, rupture uterus, acute fatty liver, acute polyhdramnios
	☆ Masses	- Complicated ovarian (ruptured CL cyst or TL cyst of V.mole) - Complicated fibroid (red degeneration)
❷	Urinary	Cystitis, pyelonephritis, stones (renal colic)
❸	GIT	Gastroenteritis, viral hepatitis, food poisoning
❹	Surgical	Acute appendicitis, acute cholecystitis, perforated DU
❺	Medical	DKA, sickle cell crisis, acute porphyria, mesenteric vasc. occlusion